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ANATOMICAL AND PHYSIOLOGICAL EFFECTS OF RAPID DECELERATION

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THE OHIO STATE UNIVERSITY
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WRIGHT AIR DEVELOPMENT CENTER
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PURFUURD

The research reports here it was carried out under Contract No. AF 33(05d)-19727, natween the Ohio State University and the Acro Medical Laboratory, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio. Capt. Ira Barash served as the Project Engineer for the Aero Medical Laboratory under the authority of Project No. 7218-71723 "Biophysics of Escape".

All animals used in the physiological experiments described in this report were treated in accordance with the rules of the American Medical Association.

ABSTRACT

The advent of modern high speed transportation has brought into extensive use many types of restraining devices as an active means of preventing bodily injury from high peak decalerative forces of short duration. Man's interest in travel in the upper atmosphere and in space itself with reasonable assurance of aftereturn has introduced, also, problems in which the tolerance of the body, anatomical as well as physiological, must be known.

This investigation has been undertaken primarily in response to the request of safety engineers who are in need of information concerning the strength of the human skeleton and the effects of rapid deceleration produced by safety devices.

Our approach to the investigation of morphological damage and the force required to produce it has been done by testing the strength of the structures that comprise the pelvic girdle inclusive of the acetabulae and femura. This has been done by analysis of the ultimate breaking strength of bones in the isolated and intact state, and by analysis of the ultimate breaking strength of a series of bones, ligaments, and muscles which comprise the joint. Tests have been conducted in both static and dynamic conditions on unembalmed human cadaver materials.

Detailed methods of applying force and resulting fractures have been described for all structures. In the intact condition, fractures of the femure do not occur when the force is applied by cables to the inferior surface of the neck of the femur, or when a force is applied. to the base of the sacrum. Fractures of the lateral wall of the pelvis and acetabulum do not produce fractures or disjunction of the sacroiliac joint. The strength of the pelvic ring is directly related to the strength of the unterior and posterior wall and as such the strength of these walls must be known to correctly ascertain the inherent strength of the entire pelvic ring. Primary fractures of the publicrami, unilateral or bilateral, produce secondary fractures or disjunction of the sacro-iliac joint by disrupting the integrity of the counter-arch of the pelvis. The counter-arch is, therefore, the important factor in maintaining the inherent strength of the pelvis for the reason that it ties anteriorly the weight-bearing arches that are confluent with the sacrum.

Previous data from this laboratory (Joffee 108) shows that congestion of the lungs interpreted as congestive heart failure, is the primary pathological result of deceleration into abdominal safety belts. By high speed X-rays taken at the rate of 50 frames per second at the instant of deceleration, we have described a possible mechanism for the cardiac failure produced by Joffee.

These experiments suggest that the mechanism by which cardiac damage is produced in rapid deceleration involved the action of the belt in suddenly blocking blood flow in the abdomen, both in the vena cava and the abdominal aorta. Since the flow of blood from the upper region of the body is toward the heart, the effect produced in similar to that of a closed liquid system subjected to a force from below and above. Therefore, the heart is squeezed between these two forces and stretched in the transverse direction. This phenomenon is analogous to the deformation of a rubber balloon filled with water which is suddenly and forcibly deformed by forces acting from below and above. In these experiments, the changes noted either diminished or completely disappeared within a fraction of a second after the force was applied. Even so, it is possible that this force might produce cardiac pathology by momentarily stretching the muscle beyond its limit of distensibility. Furthermore, it is likely that higher forces of deceleration might result in permanent deformation of myocardium.

A third phase of investigation was undertaken to determine the tolerance of dogs to rapid deceleration with force applied to the region of the ribs by abdominal safety belts. Final conclusions on this phase must be deferred because it has not been possible with the existing apparatus to deliver enough force under free fall conditions to produce permanent damage to the skeleton or visceral organs.

Nevertheless, some minor but definite damage was noted to the heart, liver, and kidney at 56 g, measured by accelerometers at level of the fourth rib. Histological examination of these organs revealed hemorrhage in myocardium, subcapsular hemorrhage of liver, and in one case, a general congestion of the vessels of the cortex involving the interlobar vessels.

Though the heart, by virtue of its position, is particularly vulnerable to violence, the liver and kidney may also be included as areas in which damage may be produced.

PUBLICATION REVIEW

This report has been reviewed and is approved

FOR THE COMMANDER:

JACK BOLLERUD

Colonel, USAF (MC)

Chief, Aero Medical Laboratory Directorate of Research

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SECTION I

INTRODUCTION

For many years the safety engineer has been interested in the problem of preventing bodily injury from rapid decelerative forces. With the advent of modern, high-speed transportation this problem has become extremely important, not only to the aviator, but also to the automobilist, and even to the pedestrian. If man is to successfully travel in the upper atmosphere and even in space itself with reasonable assurance of safe return, he must have a thorough knowledge of the forces he will encounter and his tolerance to these forces. Furthermore, the craft in which he travels must be engineered to his physiological and anatomical tolerance.

One may actively protect himself by keeping these decelerative forces within the physiological and anatomical safe limit, but unfortunately this safe limit has not been accurately ascertained. Added protection may be gained by the adoption of various types of devices such as the anti-g suit, or by devices such as the abdominal and/or shoulder type safety belt. It is the latter, the safety belt, that is of particular interest in this research. The effects of safety belts have been studied with consideration of both the anatomical and physiological aspects. This combination of anatomy and physiology should at once emphasize the complexity of the problem of tolerance and should emphasize the numerous aspects which one must, of necessity, consider in solving the problem of "how much force can the human body tolerate." At a recent meeting of the Anatomists, the fact was stressed that the solution of anatomical problems must include the problem of function; the converse is also true -- "the study of function must also include the study of structure." In this research, we have a striking example of the common meeting ground of both.

The anatomical aspects have been visualized from the mechanical viewpoint, i.e., a survey of the component parts of the skeleton receiving the force and the magnitude of the force required to cause complete fracture of the structure. This, in turn, was approached from the standpoint of first isolating these structures, testing each separately and, secondly, testing the same structure in its true anatomical orientation.

We have also studied the thoracic and abdominal viscera from the functional viewpoint since, during the early stages of this research, it became increasingly evident that the heart was extremely vulnerable to nonpenetrating forces which strike the chest or to forces which were delivered to the

chest by way of the abdomen, such as happens with the abdominal type safety belt. Occasionally, following accidents which involve application of severe forces to the thorax of a person, a clinical condition of congestive heart failure is observed. Our interest is in describing the mechanism which produces this symptom in controlled laboratory experiments.

This line of research was first adopted because the original sponsor of the project, the National Safety Council, was confronted with industrial accidents directly a lated to safety belts and harnesses in which individuals have sustained severe injury and even death. The symptoms, in most cases, were latent in appearance and poorly defined. The cause of injury or death was immediately attributed to the attachment of the safety belt to the inferior surface of the chondrocostal arch which resulted in the compression of the abdomen and in forcing the viscera against the diaphragm.

This research was later taken over by the Wright-Patterson Air Force Base because of their interest in the problems of the strength of the human body in relation to both the ejection seat and the safety belt.

One might assume that certain injurious effects, both physiological and anatomical, are brought about by the use of safety belts when individuals are subjected to high-peak decelerative forces of short duration. However, at least one group presently investigating the problem has minimized, and in some instances completely denied, any effects of forces applied in this manner. A cursory glance at the literature and experimental results of the various groups investigating this problem will emphasize its controversial nature. Much of this controversy has come about as the result of crash injury investigations in which hundreds of accidents were analyzed from the standpoint of location of injury and comparison of the injury related to the use of, or disuse of , the lap belt and/or shoulder type of safety harness. It is the opinion of Hugh DeHaven, head of the National Research Council on Crash Injury Research, that seldom, if ever, has the use of the safety harness resulted in injury in the region of the body where the belt is attached and hence, the head is the most important region of injury. This is due to the head striking some hard object. Safety, therefore, can be engineered since the human body has a tolerance considerably in excess of the strength of the surrounding structures.

We are in complete agreement with the proposition that the human body can withstand extremely high-peak forces of short duration. We must, however, accept with certain reservations, the proposition that safety belts are harmless. These reservations are based on evidence from this and other laboratories and from considerable evidence gained

chest injuries produce symptoms of cardiac failure, namely, the symptoms of congestive heart failure. It is not our desire to completely disagree with DeHaven and certainly not to recommend the disuse of belts. We believe, as he must believe, that some injury to the viscera and bones is always believe, than loss of life which would result if the belt were not used. We believe, nevertheless, that in order to provide complete protection the latent symptoms which result from impact force, either with or without harness, must be fully recognized and further engineering must be based upon this recognition.

An example of the opposite point of view is the crash injury report of Dr. Teare which appeared in the BRITISH MEDICAL JOURNAL in September, 1951, concerning the Viking aircraft crash at London Airport. We wish to quote from the article appearing in SCIENTIFIC AMERICAN, December, 1951, as reprinted by the Crash Injury Research Council in order to elucidate the controversy more clearly.

"When an airplane crashes, the safety belt that passengers are required to-fasten around their waists may become a deadly hazard. A British physician, named Donald Teare, examined 28 victims of a crash at London Airport and found that 16 were killed by chest and abdomen injuries resulting from acute flexion of the body over the safety belt. Eight of the victims had suffered a rupture of the aorta. An extremely rare injury, almost never found except in plane accidents."

Equally controversial is the question of man's tolerance to high-peak impact forces. It has been shown by data from crash injury in aviation and in free-fall from high altitudes that under certain conditions, the human body can tolerate 165 g's without serious damage. On the other hand, it was the popular belief that the snubbing action of a 1000-lb. safety belt was capable of producing considerable internal injury and was even capable of cutting people in two.

Obvious difficulties arise when one attempts to understand the tolerance of the human body to high-impact forces since, as one would naturally expect, human volunteers are hard to obtain in this hazard-ous work. This is the approach to the problem in the investigation at Muroc Lake, California, at the Edwards Air Base. To date, humans have been decelerated at a peak of 46 g's without apparent injury. In the absence of human volunteers, however, one must accept the line of investigation adopted by the Committee on Crash Injury Research which attempts to reconstruct accidents by a determination of the average "g" force from the decelerative distance and the velocity at impact. This procedure does not, however, seem to solve the general

problem because the phenomena are not controllable and only a deduction about the actual forces involved can be made. One may, on the other hand, proceed as we have done, using unembalmed human cadaver material and experimental animals in laboratory controlled experiments. With Crash Injury, as with our own experiments, difficulties arise when one attempts to transfer these data to human living conditions. We feel that unembalmed cadavers approach the living state as nearly as is practicable at this time.

Four important factors must be controlled: (1) magnitude of the force; (2) direction of the applied force; (3) duration of the force and (4) the area of the body over which the force is applied. It seems altogether believable at this point that a tolerance exists for each of the vectors resulting from the myriad positions in which the body may be placed and also for the infinite rates of deceleration which exist. It is for these many reasons that when one speaks of tolerance, the tolerance to each of the above variables must also be given.

No one laboratory is capable of providing all the data which is necessary to fully understand the resistance of the body to deceleration, or to provide an anlaysis of all the forces involved. The ultimate goal of the various laboratories engaged in this work is to provide complete protection to individuals subjected to high decelerative forces. Much of this data, especially that obtained from bone studies, has been used to construct a model of the human body which may be used in test equipment rather than using the living human body itself.

We have pointed out that the approaches to the subject of tolerance of the human body and protective devices against high-peak decelerative forces are many. Since this type of investigation can be considered to be in its infancy, we have tried at this stage, to cut down trees rather than bushes. This report contains several aspects of the various approaches to this problem. The three-fold objectives are as follows:

- 1. To provide information concerning the strength of the pelvic walls, inclusive of the acetabulae and femurs.
- 2. Through the use of high-speed X-ray, to describe a possible mechanism for the production of cardiac failure.
- 3. To measure the tolerance of dogs to rapid deceleration and to provide information which would contribute to the solution of the problem concerning the possible danger resulting from the use of abdominal safety belts in rapid deceleration.

SECTION II

REVIEW OF LITERATURE

A. Nonpenetrating Chest Injury

In this laboratory it was shown that the cardiac failure produced by high-peak impact was associated with the symptoms of nonpenetrating chest injury. Since the literature on cardiac failure as a result of deceleration into abdominal safety belts is practically nil, it is advisable to give a review of the literature on nonpenetrating chest injury; first, because the electrocardiographic changes under the conditions of our experiments were shown to be strikingly similar, and secondly, it seemed important from the standpoint of determining and understanding the mechanism for the production of such injury.

The legal aspect of the problem has great importance in determining whether or not a given individual should receive compensation. This question alone has given impetus to the little experimental work that has been done in this field. Heacht (lla) points out its importance from both the industrial and military points of view. Most papers, however, stressed to some degree its importance in determining compensation for individuals who had received some degree of non-penetrating chest injury. In this age of rapidly moving forms of transportation in which individuals may be subjected to nonpenetrating chest injuries by a variety of means, the importance of this problem in protecting them financially and from the possibility of heart damage is readily apparent.

Misinterpretation and misgiving concerning the degree of cardiac damage from nonpenetrating wounds is obvious from a cursory glance at the literature, since most of the evidence compiled is from the clinic. For example, Bright and Beck (10) made a study of some 200 cases in which individuals were known to have sustained some degree of cardiac injury from nonpenetrating chest wounds. About 90% of these cases resulted in fatal heart rupture and the obvious conclusion from this statistical analysis showed the heart to be capable of withstanding only a small degree of violence and that recovery is the exception and not the general rule. These authors, however, were quick to point out that the majority of nonpenetrating wounds are not recognized clinically, since the degree of damage may be subtle or latent in appearance; therefore, the exceptions more often found their way into medical literature. The latent appearance of damage is brought out by Barber (4) who reported a case of cardiac injury dating back to 1917.

At this point, the major problem involved may be stated in the interest of clarity. Kissane, et al, has made this clear: "That severe trauma (nonpenetrating) to the chest will cause lesions of the heart, great vessels, and pericardium is well substantiated in medical literature from autopsy reports. However, it is not generally accepted that less severe trauma will cause mild changes which have a tendency to recover." Addit that papers may be cite? The further elucidate the problem. Sigher (20) states: "Frauma and is frequently overlooked because of the feeling that the chest will states, "opinions differ pertaining to production of cardiac lesions by nonpenetrating wounds of the chest," pointing out that these studies lack information concerning normal conditions of the heart prior to injury.

Since for proper evaluation the method used in collecting this data is important, the two methods will be described briefly. Experimentally, dogs have been used and subjected to varying degrees of chest injury. The results were recorded on electrocardiographic machines and collected from gross and microscopic autopsy findings. Because of the uncertainty with which these data could be applied to human beings, attempts were made to gather clinical cases as a result of industrial and automobile accidents in which it seemed likely that the individuals had received some degree of chest injury. Both methods are subject to criticism.

The types of forces which cause cardiac damage were analyzed and are here presented:

- 1. Blows to the precordium (lla, 26, 34, 35)
- 2. Lifting of heavy objects (24)
- 3. Sudden compression of the abdomen. This occurs with abdominal safety belts (19, 110)
- 4. Compression of the chest (24)
- 5. Sudden increase in intra-abdominal pressure (24)
- 6. Injuries distant from the heart may also cause damage; sudden compression of the legs and abdomen (10, 31)

Although the report of Bright and Beck (10) does not deal directly with nonpenetrating chest wounds, this paper has been reviewed because it elucidates one mechanism, or a possible mechanism of producing heart damage.

Forces cause damage by:

- 1. Throwing the heart against the bony structure of the thoracic cage (18, 19):
- This causes the heart to be torn loose from its moorings to the pericardium and the great vessels (19);
- 3. Forcing the blood back to the ventricle causing an increase in intracardial pressure (7, 19);
- 4. Causing a change in the volume and shape of the chest and a consequent displacement of the heart and mediastinum (19).

Upon receipt of injury:

- 1. The heart may rupture or burst by being thrown or compressed against the sternum or vertebras (7);
- 2. The myocardium may rupture (8);
- 3. Contusion may occur with a subsequent softening of the myocardial tissue. Rupture may occur later (8);
- 4. If the damage is not too severe, the heart may recover (34, 35);
- 5. Symptoms and electrocardiographic changes may appear at a later date (110, 11a);
- 6. Heart may fail completely (34, 35);
- 7. Symptoms may persist and be accentuated by vigorous exercise (10).

Since the same type of force does not always produce the same type of damage, certain factors have been listed that slter the nature of the force:

- 1. Flexibility or resiliency of the thoracic cage. Young people seem to be less susceptible to damage to the same force.
- 2. Condition of the heart prior to application of force. Diseased hearts are more susceptible to the same force (24, 25, 31).

- 3. Vago-sympathetic imbalance (34, 35).
- 4. The phase of the respiratory cycle. Prior warning of the impinging force causes the individual to close the glottis and be in deep inspiration with the result that the force is transmitted directly to the thoracic contents instead of being damped (18).
- 5. The phase of the cardiac cycle. The heart in systole is more vulnerable to damage than is the heart to diastole (18).

To date, no objective symptoms referable to heart damage are known, and it is for this reason that many cases of nonpenetrating wounds do not find their way into the medical literature. However, a list of clinical symptoms has been compiled and listed:

- 1. Irregular heart sounds
- 2. Slight dyspnea.
- 3. Gallop rhythm.
- 4. Systolic murmurs.
- 5. Pericardial friction rub.

Barber (4) has made a list of symptoms of long duration, whereas the above are usually found to be transient:

- 1. Partial heart block.
- 2. Auricular fibrillation.
- 3. A picture of coronary thrombosis.

The majority of damage seems to be confined to the myocardium and the pericardium (26, 34, 31, 8). Ruptures of the valves occur to a lesser degree. Kissane states (35): "A study of clinical cases showed that the most frequent injuries were noted in the pericardium and myocardium and related to the degree of external violence. In one case a rupture of the valves was noted."

The electrocardiographic picture is suggestive of experimental coronary artery occlusion and the disease, pericarditis. This was emphatically brought out by the work of Randles (36).

It has already been mentioned that the changes in the heart do not appear immediately; therefore, the electrocardiograph provides a

ready means of diagnosing injury if frequent records are made following injury to the chest. In cases where only one lead was taken, it was confined to the more diagnostic lead II. Where the course of cardiac injury was followed over a period of time, the three standard leads were taken. However, a very important fact was brought out by Hecht (lla); he stressed the need for the precordial lead in addition to the others since in his study of a clinical case, changes were noted only in precordial leads.

It has been definitely established that these changes are those associated with coronary occlusion and pericarditis (4, 8, 24, 34, 26), and involve changes in the QRST complex. Slurring of the R wave, temporary changes in the direction of the wave, voltage changes in the QRS, and axis deviation have also been noted. Other changes have been shown, but the list above is considered to be the more important. Evidence indicates that these changes appear from time to time and that no two cases are exactly alike.

Perhaps the main criticism of all the experimental work lies in the fact that in all but one case, there was no attempt to calculate the magnitude of the force. If, as Sigler (24, 25) points out, the velocity of the force as it strikes the body is important, then knowledge of the forces involved is ever important. The Randolph Field report represents the first attempt to do this. However, their work may be criticized from the point of view that the force which they used (1000 g's) is too great, especially if one attempts to show which regions of the body are more vulnerable to force as compared with other regions — in other words, to find the weakest link in the chain. This report, which assumes that the vagus is the mediator by means of which disturbances are conducted, cannot stand up to a critical statistical analysis.

Much evidence was obtained from clinical cases which could be analyzed only after the accident occurred. Leinoff (19) points out: "The history of the heart is the most important factor in determining the relation and subsequent degree of cardiac disability resulting from nonpenetrating chest injury." Obviously, this was not possible in the majority of cases.

The lack of carefully controlled experiments is evident. More experiments of the type performed by Kissane (34, 35) and his group are necessary before any definite statements can be made concerning the problem.

Though the problem of nonpenetrating chest wounds is far from solved, nevertheless certain conclusions can be drawn from the existing information:

- 1. Trauma to the heart is a frequent occurrance in bodily injury.
- 2. The force necessary to damage the heart need not be severe.
- 3. Trauma to the heart may result in recovery in one case and prove fatal in others. Factors which alter the nature of the impinging force have been listed.
- 4. Recovery from trauma is the general rule and not the exception.
- 5. Diseased heart is more vulnerable to damage from an impinging force than is a normal heart.
- 6. The mechanism in producing heart damage is not definitely known.
- 7. Electrocardiographic changes involve the QRST complex and give the picture associated with coronary artery occlusion and pericarditis.
- 8. Electrocardiographic changes may be transient and latent in appearance; therefore, frequent records should be taken following violence to the chest.

B. Mechanical Properties of Bones

Until the past several years, the literature concerning the mechanical properties of bones was indeed scarce, in spite of the fact that its origin dates back to 1638 when Galileo Galelei studied the mechanical properties of bones in conjunction with studies on beam mechanics.

It was not, however, until several hundred years later that studies of any import were undertaken. In 1867 Von Meyer made several studies concerning the internal architecture of the femur and attempted to correlate this information with the theoretical stress-analysis studies of the brilliant mathematician, Culman, who described the trabeculae arrangement of the femur. (59)

This theoretical approach gave way about ten years later, 1876 and 1880, when Rauber and Messerer investigated the mechanical properties of the femur by placing samples in a testing machine and subjecting them to static loading to their ultimate breaking strength. (66, 72)

In 1892, Wolff conducted investigations on the functional forms of bones and formulated what is known as WOLFF'S LAW, a theory which is still contested, but which receives support from many investigators today (76a). According to Wolff, the internal structure of bone is influenced directly by its external loading.

A rather extensive review of the literature up to 1917 was given by Koch (59) who combined the literature of the past with his own original ideas into a theoretical analysis of stress distribution of the femur under vertical loading. He, in addition, calculated the stress throughout various regions of the femur as 100 lbs. from a graphical analysis of cross section.

The difficulties encountered in bone studies were apparent to even these early investigators. Attempts were made to overcome these difficulties by the construction of models and thereby to reduce the heterogenous construction of bone to the homogeneous structure of models. The first of such models was constructed in 1885 by Roux who loaded paraffin coated rubber models and deduced mechanical properties of bones from these models.

No work of any consequence was reported until 1940, when Milch (67) conducted further investigation along the line of Roux by the construction of models from polymerized phenyl formaldehyde resin. By photoelastic studies, he deduced properties which he thought characteristic of bone itself. The difficulties here are obvious: first, can the homogeneous characteristic of the model be directly applied to the heterogenous characteristics of the bone structure and secondly, can the dimensional pattern of the model be, with impunity, directly applied to the dimensional characteristics of the bone?

We must, however, mention the work of Kuntscher in 1935 and 1936 since it introduces a new method for the study of stress analysis. This involves an extension of the work of Roux. In this case, bones were coated with a low tensile strength material, colophonium, which, when stretched beyond its elastic limit, caused cracks to appear in the region surjected to the highest tensile strain.

During World War II, the Germans recognized the need for this type of information. Their experience led them to believe that the spinal column seemed to be a critical area as a result of deceleration from

the ejection seat. This led to an important investigation of the tolerance of the spinal column in both the cadaver and living human subjects. This involved a study of the structures of the spinal column in the isolated and the intact state. The strength of the individual vertebrae of the spinal column is listed below:

SEGMENT OF SPINE	BREAKING LOAD (Kg)
C-4	275
T-1	450
т-6	600
T-8	600
T-9	720
T-10	770
T-11	860
T-12	800
L-1	1000
L-2	830
L-3	940
L=4	900
L-5	1000

The following data were obtained from a study of the tolerance of vertebral complexes to static loading:

					T-10 to L-3
T-12	•	•	٠	•	690 Kg
T-1) T-2)	•	•	•	•	840 Kg
7 vertebral segments T-8					T-7 to L-1 540 Kg

The dynamic tolerance of the body was directly related to the tolerance of the spinal column. Experiments on human living subjects place this tolerance at 26 g if the force acts for 0.0005 seconds.

The author suggests that in an entire centrally loaded spinal column the weakest link in the vertebral chain, based upon strength of and the percentage of the load carried by the individual vertebra, L-1 would collapse after the absorption of approximately 10 m/kg of energy.

More recent work at Wayne University on the study of bone structure and fracture locations uses the coating of bones with "stress coat" and applying both static and dynamic loads to these isolated structures. Early work on the skull by "stress coat", which has a tensile stress of 0.00085 inches/inch, and by strain

gauges, shows the skull to deform when 615 inch pounds of energy is absorbed for a period of 0.0012 seconds. This same group has conducted studies on the femur and pelvic girdle.

Another group of investigators at Naval Medical Institute, Bethesda, Maryland, has appared the mechanical properties by studying strength per unit a confidence. Samples of bone from various regions were milled down to desired size and them subjected to both tensile and compressive strain.

The data on bone samples obtained from femur, tibia, fibula is as follows:

Studies to determine the effects of embalming showed increase in strength from 6 to 116%.

Lastly, the work done in this laboratory (48) (108) on the strength of bones has been approached by testing the ultimate breaking strength of the structures comprising both the pelvic girdle and the pectoral girdle.

Control of the Contro

C. General Aspects of Deceleration

Considerable information concerning the tolerance of the human body to rapid deceleration has been obtained from Crash Injury Research and experiments on human volunteers at Muroc Lake, California. Crash Injury Research involves an analysis and reconstruction of accidents, both civilian and military. From these investigations, knowledge of the tolerance is based upon the known ultimate breaking strength of the safety belt which broke during deceleration, but which produced little or no damage to the subject. On the basis of this information, the strength of safety belts has been constructed to withstand forces as high as 9000 lbs.

According to the Committee on Crash Injury Research, it has been shown that individuals have survived accidents from free fall drops as high as 200 feet resulting in an estimated 200 g force.

Perhaps the most significant research from the standpoint of human tolerance to linear deceleration, is in progress at Muroc Lake, California, under the direction of Lt. Col. Stapp (152) (116). To date, 53 experiments have been conducted on live human subjects facing forward on a rocket propelled sled. Range of deceleration on the sled ranged from 10 g at 757 g/second to 46 g at 500 g/second. Duration of deceleration ranged from 0.15 to 0.35 seconds.

The degree of voluntary tolerance was apparently related to the type of restraint applied to the subject. With standard Air Force harness, this tolerance was approached at 17 g at 1000 g/second. With 'V' leg straps and shoulder straps, the voluntary tolerance was increased to 46 g with rate of deceleration at 500 g/second.

SECTION III

ANATOMICAL DESCRIPTION.

In this investigation we are interested in the more extensive problem of general safety, which we have attacked in part by an analysis of the ultimate breaking strength of bones, isolated and in intact state and the ultimate breaking strength of a series of bones, ligaments, and the muscles which comprise the joint.

Nature has assigned bones two very important functions: First, the function of supporting the softer tissues which surround them, and in some cases forming a protective box or cage for these organs such as the brain in the skull, the heart and lungs in the resilient thoracic cage, or the spinal cord in the backbone. If one is to understand the body in terms of its tolerance to force suddenly and violently applied, some knowledge of the strength of these protective cages is mandatory. Secondly, bones act as levers in the performance of movement and as such transmit both tensile and compressive stresses. This function is obviously performed with negligible deformation to the bone itself. Then same strasses can, however, be produced in a variety of other ways - intentionally, as in the case of the ejection seat, parachute descent or safety belt and accidentally, as in the case of automobile or airplane crashes. If one is to engineer against such stresses, knowledge of the structure in terms of its ultimate strength is again mandatory.

Bone is made up of 69% inorganic matter which is mostly calcium, and phosphate, and 31% organic matter which is mostly fine fibers forming inter-lacing bundles, and connective tissue cells and bone corpuscles placed between these bundles. It is this combination of inorganic and organic matter, together with the architectural arrangement of these elements, that makes the bone not only a hard material but also a structure resistant to both tensile and compressive stresses. The brittle and weak bones of old age are due mostly to a diminution of fibrous tissue and not to any change in the content of inorganic matter. Joffee (108), in this laboratory, showed that the strength of ribs could in no way be related to bone ash content.

Bone may be either compact or spongy. The compact bone forms the outer layer which is immediately surrounded by the periosteum. It is this layer which gives bone its inherent strength and it may be said that the strength of a bone is directly proportional to the thickness of this layer. The inner spongy layer is somewhat hard, but is extremely weak. Data on shoulder girdle shows the strength of bone can be related to thickness of the outer compact layer. Bone seems well adapted to the job which it has to perform. This it does with both strength and ecomomy. The usual combination of organic and inorganic materials has already been mentioned with relation to its strength. The mechanical arrangement of the tubular lamellae also provides a structure capable of resisting bending in either direction. Koch and others have analyzed the femur from a mechanical standpoint and came to the conclusion that the tubular lamellae had the best possible arrangement for resisting both tensile and compressive forces. As they approach the upper end of the femur, tubular lamellae leave the compact bone, arching and reaching the head of the femur and greater trochanter. This system interposed between a second system which springs from the greater trochanter and head of the femur, crossing the former system at right angles forming a firm, rigid and inherently strong structural member.

Of special interest in this phase of the study of the tolerance of the human body to rapid deceleration are the bones which comprise the hip which is also called the pelvic girdle. Also of interest is the femur which articulates with the pelvic girdle via the acctabulum. All these structures are important when any force is delivered from below, that is, to the inferior surface of the neck of the femur or to the sacrum.

The first of the three bones comprising the hip bone is the ileum, the largest and the broadest of the three. The second is the ischium, which is broad and thick behind the acetabulum, forming a flat bar of bone, the lower boundary of the obturator formen and projects to the pubic symphysis as the inferior rami. The third bone, the pubis, completes the boundary of the obturator forming the superior rami. An analysis of these structures is important when one considers a force delivered to the body of the symphysis itself. The conjoined rami of the ischium and the pubis of the two sides form the pubic arch in front.

The bones of the peivic girdle can be divided into three walls; the posterior, the anterior, and the lateral. The greater part of the posterior wall is formed by the sacrum and, by means of the sacro-iliac joint, this segment is firmly wedged between the medial curved elements of the ilia. By virtue of the interlocking character of this articulation, together with the interposed short and taut anterior and posterior transverse ligaments, it possesses great inherent

strength. It is this joint which carries the greater part of the weight of the upper part of the body. It is held in place by these broad ligaments and thus, results in very little or no movement of this joint. Through this joint both pairs of pelvic arches become confluent with the sacrum. For this reason alone, this is one of the most significant and important articulations. Inside the sacrum is a canal containing roots of the sacral and coccygeal nerves, filum terminals and other important nerves. Because it contains these important structures and because it is situated in a strategically important position to support the upper frame, the ultimate strength of this joint is exceedingly important in the tolerance of the body to high impact g force.

It has been mentioned that the immominate bone is made up of three distinct bones which fuse at the region of the acetabulum. This fact is important when one attempts to analyze the nature of the fracture from a force delivered medially to the acetabulum, via the greater trochanter of the femur. The large part of the lateral wall enters into the formation of the acetabulum, the cup-shaped socket that received the head of the femur. The upper part of the socket is directly related to the femore-sacral arch. The right and left sides of the innominate bone articulate anteriorly to form the public symphysis and posteriorly with the articulating surfaces of the sacrum, form the sacro-iliac joint.

The third wall, the anterior wall, of the pelvic ring is formed by the union of the bodies of the pubic bones from which rami, the superior and inferior rami, extend laterally and backward uniting with the ilium and the ischium of the lateral pelvic walls. The anterior part of the pubic arch may be considered as a counterarch or tie that unites the weight-bearing arches, in such capacity it is vitally important in maintaining the strength of the pelvis in its entirety.

The femur itself is probably the best known of the bones, its importance in supporting the body weight as a structural member has already been discussed. It has additional importance in that it also transmits force to the hip bone, as in a parachute descent. The femur is considered the longest, heaviest and strongest bone in the skeleton. Its upper end fits into the acetabulum and articulates with the hip joint: its lower end articulates with the tibia via the lateral and medial condyles. Both the upper and lower ends are cartilage clad and will be of some importance in testing the inherent strength of this bone. The head joins the shaft obliquely by the neck at an angle of 125 degrees near the trochanteric region. Because of this, the loading of the femur is eccentric loading in contrast to the concentric loading of a perfectly vertical structural member. The neck and trochanters, greater and lesser, joining

the shaft and neck as they do, make them extremely vulnerable to fracture. The angle with which the neck joins the head, and hence approaches the acetabulum, is important. The greater the angle the more nearly the loading would approach concentric loading and away from eccentric loading; hence the stronger the bone becomes as a structural member.

The shaft of the femur is slightly twisted and is thinnest at its middle third. The femur is firmly attached to the lateral wall of the acetabulum by a strong ligament, ligament of head of femur, attached to the pit of the head.

Some mention has already been made of the importance of ligaments in determining the strength of joints. In the region of the acetabulum, the capsular ligament with its thickened part, the ilio-femoral ligament, is attached to the lower part of the anterior superior iliac spine; the pubo-femoral ligament is attached to the lateral end of the obturator crest: the ischio-femoral ligament is attached to the ischium below the acetabulum. Of these three ligaments, the ilio-femoral ligament is the most important because it is the strongest one of the hip joint and enhances the strength of the articular capsule above. It is shaped like an inverted 'Y' whose base is attached medially to the ileum, with its apex attached laterally to the greater trochanter and the interstrochanteric line. As this ligament fans out to make the lateral attachment, a thin area is found tatween the two limbs of the 'Y'. Since the capsule extends over the entire anterior surface of the neck of the femur and is inserted along the inter-trochanteric line, the neck in this region is, therefore, included as part of the joint. Behind, the capsule includes about two-thirds of the neck and is, therefore, extra-capsular and should not be considered as a part of the joint.

The sacro-iliac joint is provided with a capsular investment, anterior and posterior sacro-iliac ligaments, which is lined by a synovial membrane. The anterior ligament is actually a broad and rather thin band which closes the joint in front throughout its entire extent. The posterior ligament is a more robust and thicker structure and is divided into three parts, the interosseous, the short posterior and the long posterior. These close the joint behind and form a dense decussation. The interosseous portion, the most directly related part of the ligament of the joint, bridges transversely across from the lateral posterior surface of the sacrum to the medial aspect of the ilium adjacent to the joint and is disposed as short isolated fascicles.

Since this research to date has considered mostly the strength of the structures which comprise the joint, a cursory survey of the classification of joints is here given. As has been mentioned, it is these joints that give continuity and maneuverability to the skeleton.

Depending upon their function, they may be generally classified as immovable or movable. Also, the chief kinds of joints have developmental differences and, on this basis, there results three main kinds of joints:

- 1. Fibrous joints which are characterized by the fact that they have either arisen in membrane, or were preceded by discrete cartilage but still have a membraneous connection
- 2. Cartilaginous joint, characterized by either continuous cartilage where cartilage has become chondrified
- 3. Synovial joint, characterized by a cavity surrounded by ligament lined with a synovial membrane which produces synovial fluid.

Of special interest is the immovable or slightly movable, sacro-iliac joint, and the synovial joint characterized by the acetabulum or hip joint which is capable of considerable movement.

The strength of the articular ligaments and muscle tension are important factors about a joint. In situations of continuous stress, such as the hip joint, these ligamentous bands, already described, are most important. They become less important in the weak and lax capsule of the shoulder joint. No ligaments, however, can resist continuous stress without support. Four factors limit movement of a joint, apposition of the soft parts, locking of the bones, tension of ligaments, and muscular tension.

During the initial phases of this research, the problem was first visualized from the point of view of the strength of the pelvic girdle by a force delivered to the inferior surface of the neck of the femur; hence, the muscles on the medial surface of the thigh (pectinous, the adductor group of muscles (longus, brevis and magnus) gracilis, and obturator externus seemed of great importance. The analysis of this medial mass of muscles was dropped when tests proved that the combined strength of both femurs, together with their architectural attachment to the pelvis, formed an inherently strong and robust joint, irrespective of the muscles which comprise the medial mass of the thigh.

ANATOMICAL

Methods and Results

In order to determine the strength of the pelvic girdle, tests were conducted upon the isolated structures of the pelvis under static and dynamic conditions. Preliminary tests were first conducted on the sacro-iliac joint since, by virtue of its position and function, it is one of the most important structures in this region.

Gradually applied forces were delivered to the sacro-iliac joint by means of the Olsen Lever Transmitting Machine. The pelvis was first denuded of all the muscles and the vertebral column was cut between the lumbar vertebrae 3 or 4. This was done to make possible the delivery of gradually applied forces to the joint without contact with the crests of the iliac. The structure was then oriented in a cement mold and the cement allowed to gain maximum tensile and compressive strength by curing under high humidity. Bone samples were also kept moist until the time of testing. A 3 x 3 x 1 inch steel block was then placed over the top of the vertebral segment to permit the delivery of force evenly throughout the entire surface. Reference of Figure 1 will make clear the technique involved.

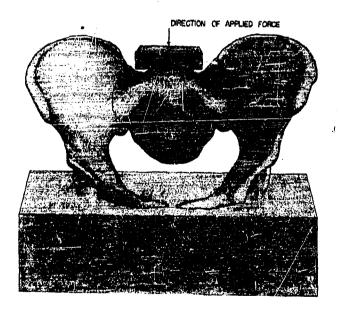


Figure 1. Mounting of pelvis for static loading showing orientation of denuded pelvis for static testing of sacroiliac joint with Olsen Lever Transmitting Machine

Specimens were taken from robust male cadavers whose cause of death was known. No samples were tested from cadavers who showed any previous damage to bone tissue.

JOINT ULTIMATE BREAKING STRENGTH (STATIC LOAD)
Sacro-iliac Average 1925 lbs.

The fracture produced under these conditions does not disrupt the sacro-iliac joint at its articulation with the ilia. The bones of the posterior region are involved; that is, the sacral cornu around each end of the sacral canal moves laterally from the lower end of the sacral canal and splits the vertebral segments of the sacrum on the dorsal surface along the middle of the spinous tubercles. This fracture occurred in all the specimens tested under static conditions.

This fracture is quite understandable in view of the rigid mounting of the pelvis in the cement mold, since a force now applied to the sacro-iliac joint at the level of the lumbar vertebrae creates not only a vector in the vertical direction, but also a horizontal vector causing a deformation in this direction with consequent elongation in the anterior-posterior pelvic plane. One must then visualize the resultant vector as an oblique vector which acts in the direction of the sacral cornu since it is this lower edge of the projections where the fracture first occurs. This type of fracture is illustrated in Figure 2.

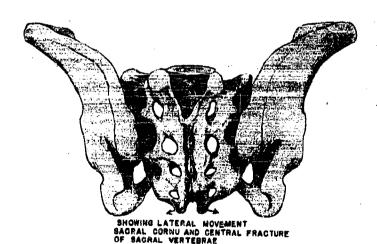
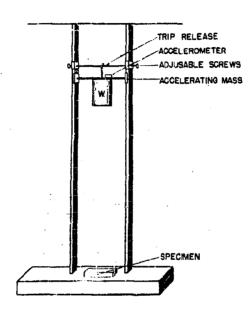


Figure 2. Posterior view of the pelvis showing fracture under static loading.

The situation is considerably changed when the force is a dynamic one. Samples were again mounted in a cement mold as described above. Forces were dynamically applied to the sacro-iliac joint by means of a pile driver (Figure 3).



ratus used for the delivery of dynamic force to structures of pelvis.

By this means a mass of known weight was accelerated a known distance. The apparatus was especially constructed to deliver a mass in free fall (except for the amount of friction due to the guide beams) with a means for changing the accelerating mass and height of fall. The guide also made possible the delivery of force at precisely the desired point. A strain gauge (Statham) type accelerometer was then rigidly mounted to the accelerating mass and the force in g's was then determined by oscillographic recording. From the formula: F = M A

Where F = force in pounds,

M = accelerating mass in pounds,

A = acceleration in terms of g's

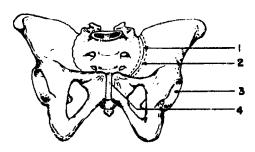
the force in pounds necessary to cause fracture or disjunction of this joint was calculated.

This type of force produces a rupture of the sacro-iliac joint proper at its articulation with the ilia of both sides. As was expected, no fracture of the vertebral segments of the sacrum itself was seen; rather, a disruption of the ligaments, both anterior and posterior short and long ligaments occurred. Again, as was expected, the ligaments themselves were not involved, but in each case the ligaments were torn from their cartilaginous mooring to the sacrum. This seems to indicate, as was also found in the case of the coraco-acromial ligament of the shoulder joint, that ligaments are extremely rugged and are of considerable strength.

JOINT ULTIMATE BREAKING STRENGTH (DYNAMIC)
Sacro-iliac joint 270 lbs.

Since this sample had been previously embalmed and was somewhat dry, no definite conclusions can be drawn from this sample and additional investigation is indicated to accurately ascertain the ultimate breaking strength of this joint under dynamic conditions. It is the opinion of this investigator, however, that bilateral fracture disjunction will occur under these conditions because a suddenly and violently applied force would cause little deformation in the anterior-posterior direction and would result in one main vector, that acting principally in vertical direction. It is for this reason that the breaking load and fractures differ considerably under static and dynamic conditions.

Reference is here made to Figure 4 - No. 1 for further clarification of fracture produced.



- I. SACRO-ILIAG JOINT
- 2. ATTACHMENT OF ANTERIOR LIGAMENTS
- 3. LATERAL WALL OF ACETABULUM
- 4. SYMPHYSIS PUBIS

Figure 4. Anterior view of pelvis. Dotted lines show region of attachments of ligaments which rupture when a dynamic force is applied to sacro-iliac joint.

A second series of tests was conducted on the femur, denuded of its muscles and under static conditions. This was done by gradually applying a force to the head of the femur with the Olsen Lever Transmitting Machine. This series was undertaken since the femur was first determined to be the most important region for the type of deceleration experienced in parachute descent and from the general consideration of the standing position in which the weight of the body is transmitted, via the sacro-iliac joint, the acetabulum to the superior and lateral surface of the head of the femur.

Preliminary tests were first conducted in the same manner as by previous investigators in order to compare our data with their data.

The denuded femur was oriented in the testing machine so that a gradually applied force made contact with the head of the femur at one point on the superior surface (point loading) and at a point on both the medial and lateral epicondyles. A force of 2000 lbs. was sufficient to cause fracture when these samples were tested to destruction. The breaking strength of 2000 lbs. and location of fracture compares favorably with the results obtained by previous investigators.

This condition shows the greatest bending moment is in the region of the neck of the femur between the junction of the neck with the head of the femur and the junction of the neck with the greater and lesser trochanters. Because of the anatomical nature of the articular capsule the fracture is intracapsular anteriorly and extracapsular posterior. ly. The capsule extends over the entire surface of the neck and is inserted along the intertrochanteric line. Posteriorly the capsule covers only the medial two/thirds of the neck.

JOINT	ultimate breaking strength (static)
Femur (point loading)	2000 lbs.

Observations of the results of these preliminary tests gave information which was used as the basis for another series of tests on the femur. Flat deformed areas were noted on the cartilage-clad head and epicondyles where contact was made between the femur and the heads of the Olsen Lever Transmitting Machine. Obviously, these deformed areas absorbed some energy before the ultimate breaking strength of the femur was reached. On the basis of this data, further investigation was conducted on the denuded femur by placing cement casts on the articular surfaces of the epicondyles and the head of the femur (area loading) in order to more correctly simulate the actual body condition. This method has advantage over previous methods of determining strength in that it makes possible the delivery of force over the area of the head of the femur and epicondyles normally in

contact with the articular surfaces. No flattened out area of deformation were noted when these samples were subjected to gradually applied forces by this method; hence, no energy is absorbed.

JOINT ULTIMATE BREAKING STRENGTH (STATIC)
Femur (with cest) 1600 lbs.

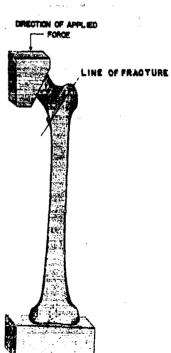
Since no energy is absorbed in deformation of the cartilage-clad articular surfaces, one would expect to find the ultimate breaking strength somewhat lower in the case of the femura subjected to gradual forces with the casts. Inspection of the above data and comparison with the breaking strength of the former shows this to be the case.

More important, however, is the difference in the nature and type of fracture under this condition. The fracture produced in the former case has already been described as being intracapsular in front and extracapsular behind. The fracture by the latter method is completely extracapsular; that is, does not involve the joint proper since it occurs laterally to the neck and carries with it part of the trochanters, and by virtue of this fact indicates also a change in the position of the greatest bending moment.

We believe at this point that the latter method herein described, approximates more closely the intact condition and described more accurately the behavior of the femur, both with respect to fracture and ultimate breaking strength.

Figures 5 and 6 will make clear the techniques used in testing this structure and the types of fractures produced.

Figure 5. Fractures of femure produced by static loading with and without casts.



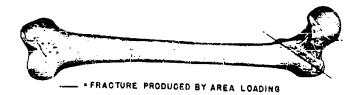


Figure 6. Method of applying static load to femurs.



A description has already been given of the nature of the sheft of the femur with particular reference to its being thinest at the middle of the shaft. This at once emphasizes a weak point in its architecture and it seemed, therefore, desirable to know its strength.

Casts were again placed over the articulating surfaces of the head, medial and lateral epicondyles of the femur and a gradual force was applied to this structure in the anterior-posterior direction at the middle of the shaft in the manner indicated in Figure 7.

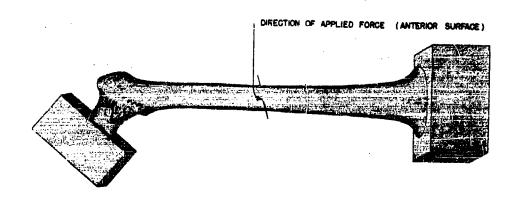


Figure 7. Method of applying load to the middle of the shaft of the femur.

1700 lbs.

A fracture, somewhat jagged in nature, occured immediately below the impinging force. This fracture was similar to that which would be obtained if a hollow wooden cylinder was subjected to a bending moment beyond its elastic limit.

Mention has been made that one of our purposes in this investigation was to contribute to the vast amount of information which is ultimately to be used in the construction of an anthropomorphic dummy. This was to be used in lieu of live human subjects in the various phases of work on deceleration where it was neither desirable nor possible to use live human subjects. With proper construction, this dummy is to exhibit the same tensile, compressive strength and the same damping effects as the human body.

In line with this long range plan, the strength of the clavicle was tested by means of the Olsen Lever Transmitting Machine and this data compared with the ultimate breaking strength of a model clavicles provided by the Wright-Patterson Air Force Base, Dayton, Ohio.

Samples were mounted in cement casts, as indicated in Figure 8, and a gradual force was applied at the junction of the medial two-thirds with the lateral one-third.

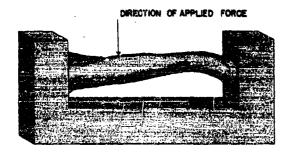


Figure 8. Method of applying a static load to the clavicles.

A fracture of the human clavicle is produced approximately 3 cm medial to the acromic-clavicular articulation. This is the area, the lateral one-third, in which the compact layer of bones becomes thin and is therefore, weak.

STRUCTURE	777 1717 3 E A O	101 5074	124 NIA C	TRENGTH	/ MM AMY/
CALCELLY AND DESIGNATION OF THE PROPERTY OF TH	11('1') M2''	H: MMH:	in inura s	COLDER BOOK COLD DE	CSTATES
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Clavicle

230 lbs.

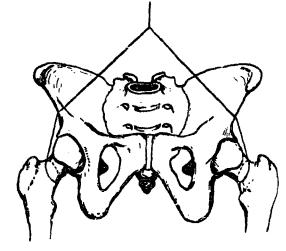
Model of clavicle

260 lbs.

Model clavicles were subjected to forces of graduated intensity by exactly the same technique. Though both models and human clavicles have almost identical breaking strength, the location of the fracture, as was expected, was very dissimilar. Model clavicles fractured at the junction of the medial two-thirds with the lateral one-third or immediately below the point of applied force. This points out one great difficulty in the construction of a model, the fact that the material for construction of a dummy is ultimately homogeneous in contrast to the heterogeneous construction of the elements of the human body.

In order to acquire preliminary data relative to the strength of the hip joint with femura attached, a series of pelves was prepared by denuding the musculature of both pelves and femura keeping intact the ligaments of the joint, the ilio-femoral, pubo-femoral, and the ischio-femoral. These ligaments constitute the chief part of the articular capsule of the joint. Since the capsule extends anteriorly over the entire surface of the neck of the femurand is inserted along the intertrochanteric line, the neck in this region must be included as a part of the joint. Behind the capsule includes about two-thirds of the neck and as a result the lateral portion of the posterior aspect of the neck is extracapsular and should not be considered as part of the joint.

The dynamic strength of the joint was investigated by application of force delivered at the inferior surface of the neck of the femurin a manner to simulate the action of a parachute harness. Figure 9 makes this method clear.



WADC TR 54-218

Figure 9. A method of applying load to the inferior surface of the neck of the femurs. The figure shows the cable attachments to the hip joint for testing the tolerance of this structure to dynamic force.

For these experiments two bodies were used. By graduated falls up to the limit of tolerance, damage was produced as follows:

In the first case (partially denuded of muscles) the joint capsule was ruptured. This occurred in the weak area of the ilio-femoral ligament. The ilio-femoral ligament, the strongest one of the hip joint, enhances the strength of the articular capsule above and is shaped like an inverted 'Y' with the apex attached to the ilium, and with base attached to the greater trochanter and the inter-trochanteric line of the femur laterally. As the ligament fans out to make the lateral attachment, a thin area is found between the two limbs of the 'Y'. This is the vulnerable place in the joint and the area that ruptured permitting the head of the femur to escape when subjected to 600 lbs. of force.

STRUCTURE	ULTIMATE BREAKING STRENGTH (DYNAMIC)
lst sample (denuded)	600 lbs.
2nd sample (completely intact)	7500 lbs.

In the second specimen (completely intact), the following results occurred by graduated free fails to the limit of tolerance:

- 1. A fracture, unilateral, through the superior ramms of the pubis, thus breaking the tie or counter arch of the femerosacral arch of the pelvis. This allowed the pelvis to spread and resulted in -
- 2. A bilateral fracture disjunction of the sacroiliac joint.
 The force necessary to cause fracture is summarized above.

Figure 10 will make clear the location of fracture involved in this method of testing the dynamic strength of the hip joint.

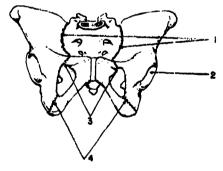


Figure 10. Bony pelvis showing regions of fracture.

- I UNILATERAL OR BILATERAL FRACTURE DISJUNCTION.
- 2. FRACTURE OF MEDIAL WALL OF ACETABULUM
- 3. FRACTURE OF SUPERIOR RANUS BILATERAL OR UNLATERAL
- 4 FRACTURE OF INFERIOR RAMUS BILATERAL OR UNIL. FRAL.
- 3 AND 4 ARE COUNTER THES OF WEIGHT BEARING
 ARCHES OF PELVIS.

The description of the pelvis showed that the anterior part of the pelvic ring is formed by the union of the bodies of the pubic bones from which rami, superior and inferior, extend laterally and backward uniting with the ilium and ischium of the lateral pelvic walls. The anterior part of the pelvis may be considered as the counter arch or tie that unites the weight bearing arches and in such a capacity, it is vitally important in maintaining the strength of the pelvis in its entirety.

Samples of pelves with soft tissue intact above the symphysis but with pelvic contents removed, were subjected to violence by means of the pile driver (Figure 3) already described. Forces of graduated intensity were applied to the pubic region in an anterior-posterior direction to the degree where fractures were induced. By virtue of the resiliency of this part of the pelvic ring, a force of 595 lbs. was required to cause fracture to this joint.

STRUCTURE	ULTIMATE BREAKING STRENGTH (DYNAMIC)
Symphysis Pubis	595 lbs.
	ومحمول والمراب

In some tests, the superior ramus fractured, and in others, both superior and inferior rami were involved in the fracture. Usually the injury was unileteral, but in one case it was bilateral and included both rami.

In these fractures, the damage was not confined altogether to the anterior wall of the pelvis, but to the posterior wall as well, i.e., the sacro-iliac joint. However, the damage to the sacro-iliac joint is a secondary involvement - fracture disjunction. The inference is that the public rami are damaged and the principal weight bearing arches having lost the tie which keeps them united, spread open and only then involves the joint of the posterior wall.

Reference to Figure 10 will make clear the location of fracture under this condition of violence.

Another series of tests were conducted on the sacro-iliac joint in order to determine whether or not a force delivered immediately to this joint would involve fractures of the femurs. It has been ascertained that violence delivered to the inferior surface of the femur (Figure 9) produces no fractures involving the femurs, but involves, rather, the weight bearing arches, since the combined strength of both femurs gives an extremely rugged and strong structural member.

With this data as background, the problem at this point may be clearly stated: "Will violence delivered to the head of the femurs,

via the sacro-iliac joint and weight bearing arches, involve fractures of the femur"?

Pelves were denuded of all muscles. The ligaments of the hip joint and sacro-iliac joint were, however, kept intact. The sample was then mounted in a cement cast up to about the distal third of the femur with the sample oriented as in standing. By means of the pile driver (Figure 3) forces of graduated intensity were delivered directly to the sacro-iliac joint to the point where fractures were induced. Inspection of the data given below shows that a force of 830 lbs is of sufficient intensity to cause fractures involving the sacro-iliac joint.

	ULTIMATE BREAKING STRENGTH (DYNAMIC)
Sacro-iliac joint	830 lbs.
The second se	

This was a bilateral disjunction, as was noted in several of the preceeding tests. No rupture of ligaments occurred. As previous tests on ligaments have shown, ligaments pull loose from their cartilagineous mooring to the skeleton.

The inference from these tests shows also that the combined strength of both fewers is stronger than the weight bearing arches.

Reference is here made to Figure 10 of the preceeding sections for the location of fractures.

The formation of the lateral wall of the pelvis and the manner in which the lateral wall enters into the formation of the acetabulum, and the cup-shaped socket that receives the head of the femur have been described. The upper part of the socket is directly related to the femoro-sacral arch and on the basis of this consideration, it is advisable in determining the ultimate strength of the structure of the pelvis to know the strength of the lateral wall of the acetabulum as well. Knowledge of strength and the location of fractures is important also when one considers that the delivery of force, such as during a fall on the side of the hip, may force the head of the femur into the pelvic cavity.

In determining the strength of this wall (Figure 4), force was applied first to the greater trochanters in the dry pelvis and later to the soft tissues overlying the greater trochanters in pelves in the recent state. The pile driver, which has already been described (Figure 3) was used to deliver forces of graduated intensity up to the limit of tolerance.

Lateral wall of acetabulum

800 lbs.

The force gradually increased to 800 lbs. was of sufficient intensity to induce fractures of the medial wall of the acetabulum and to regions of its epiphyseal junctions. Thus, the fracture may be radiating in character and accompanied by the bending in of the central part of the acetabulum. In one instance, the head of the femur was driven into the pelvic cavity acetabular fracture with central dislocations. Since none of the tests of the lateral wall destroyed the integrity of the femoro-sacral arch, though the force was delivered directly below its anterior extremity, it follows therefore, that the sacro-iliac joint was not involved in the initial injury.

Up to this point, the individual structures of the pelvis and the hip joint have been tested for their ultimate strength and for the location and extent of the resulting fractures. The existence of weight-bearing arches have already been mentioned on two occasions:

- 1. Fractures involving the sacrum with a force delivered to the inferior aspect of the head of the femur, and
- 2. Fractures involving the sacrum with a force delivered to the anterior surface of the symphysis pubis.

With this definite and important inter-relationship indicated, an additional series of tests were conducted to determine the part played by both the anterior and posterior pelvic walls on the inherent strength of the entire pelvic ring.

Pelves in this series were mounted in cement up to and including the distal third of the femurs and has been described. The viscera in the pelvis was removed, however, the muscles and ligaments were left completely intact. Forces of gradual intensity up to the limit of tolerance were delivered by means of the pile driver directly and immediately to the sacro-iliac joint in order to interrupt the strength of the posterior pelvic wall. Inspection of the data given below shows that in this case, a force of 775 lbs. was of sufficient intensity to cause a fracture disjunction of this point.

STRUCTURE	ULTIMATE BREAKING STRENGTH (DYNAMIC)
Sacro-iliac joint	775 lbs.
والمستقدم والمستقدين أراء أوالما المستقدين والمستقد والمستقد والمستقد والمستقد والمستقد والمستقد والمستقد	

This figure compares invorably with the strength of this joint as presented in the preceding section. In that particular sample a force of 830 caused a fracture disjunction of this same joint.

With the destruction of the posterior pelvic wall, and hence, an interruption of the weight-bearing arches, a force was then delivered directly and suddenly to the symphysis pubis, which it must be recalled, is the union of the conjointed rami, i.e., the union of the superior and inferior rami anteriorly. A force of 350 lbs. is of sufficient intensity to cause a fracture in this area.

STRUCTURE ULTIMATE BREAKING STRENGTH (DYNAMIC)

Symphysis pubis with fractured sacro-iliac joint 350 lbs.

Fractures in this case were identical in location to those found in all other cases, i.e., involving a bilateral fracture of both the superior and inferior ramus.

Having thus visualized the problem from the point of view of the support that the anterior wall gives the posterior wall, the reverse question also presented itself: "What support does the posterior wall give the anterior wall?"

Data has already been given relative to the strength of the joint but will be repeated here for purpose of clarity. Force was delivered by means of the pile driver to the anterior surface of the symphysis pubis in order to disrupt the union of the weight-bearing arches in front. Inspection of these data below shows a force of 595 lbs. was of sufficient intensity to cause a bilateral fracture involving both the superior and inferior rami.

STRUCTURE	ULTIMATE EREAKING STRENGTH (DYNAMIC)
Symphysis pubis	595 lbs.
Sacro-iliac joint	480 lbs.

After disruption of this joint, the sample was mounted in cement cast as were the preceding samples. Force was again delivered directly and suddenly by means of a pile driver to the sacro-iliac joint. Inspection of the above data shows that a force of 480 lbs. was of sufficient intensity to cause a fracture of this joint. Fractures were again identical in nature to those herein described which involved this joint. (Figure 10)

ANATOMICAL DISCUSSION AND CONCLUSIONS

Discussion

A history of the development of studies relative to the strength of bones and the methods for testing has already been reviewed. Our approach to the problem of strength of the pelvic girdle, inclusive of the acetabulae and femurs, was to determine the ultimate breaking strength of the isolated and intact series of structures that comprise the hip joint. With a knowledge of both static and dynamic tolerance of this region, forces may then be kept within the limit of tolerance in cases where human beings are willingly subjected to decelerative forces. The same information should be particularly useful in the construction of an anthropomorphic dummy to be used in experimentation where the approach to the problem by using live human individuals might subject them to unforeseen hazard. To this end, we hope we have been successful.

We have selected the approach by studying ultimate breaking strength of bones on the basis that it provided more useful information than the study of strength by "stress coat" or by milling bones to desired size and studying strength per unit volume. Though our approach has involved many difficulties, principally equipment, we believe the methods to be fundamentally sound and the results to be accurate to ± 15%.

The difficulties in obtaining cadaver materials for this type of experimentation should be obvious, especially since we have endeavored to use only recent state materials rather than ordinary Medical Dissecting Room materials.

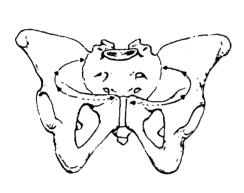
The importance of the adductor group of muscles was indicated in the early stages of this investigation for the reason that the area of the femur subjected to violence from a free fall, such as jolt received during opening of parachute, will depend upon the nature of the adductor group of muscles. From the data indicated in the series of experiments in which violence was delivered to the inferior surface of the neck of the femur and in those in which this medial mass of muscles was removed, no fractures were obtained which involved the neck of the trochanters of the femur. Fractures were obtained only in isolated tests on the femur.

Attention was, therefore, focused from the importance of this medial mass of muscles to a survey of the weight-being arches,

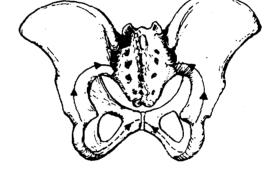
especially since fractures obtained in the completely intact specimen occurred:

- 1. As a unilateral or bilateral fracture through the superior ramus of the pubis, thus breaking the tie or counter arch of the femoro-sacral arch of the pelvis
- 2. As a bilateral fracture disjunction of the sacroiliac joint.

It is, therefore, important at this time to emphasize certain structural areas of the pelvis to which reference has already been made; namely, the weight-bearing arches, two in number and bilateral in position.



SOLID LINE - FEMORO SACRAL ARCH BROKEN LINE - COUNTER ARCH OR TIE



SOLID LINE - ISCHIO SACRAL ARCH BROKEN LINE - COUNTER ARCH OR TIE

Figure 11. Anterior View of Pelvis Showing Femoro-Sacral Arch

Figure 12. Posterior View of Pelvis Showing Ischio-Sacral Arch

One pair extends from the superior pubic rami backward and above the acetabula to the sacrum through the ilio-sacral joints. (Figure 11) This is, in fact, a bony buttress and may well be designated the femore-sacral arch and receives the weight of the body in standing.

The other pair of arches passes from the ischial tuberosities upward and behind the acetabula to the sacrum through the sacro-iliac joint. (Figure 12.) This constitutes another bony buttress the ischiosacral arch and functions in weight bearing in the sitting position. These arches are further strengthened by counter-ties; the femorosacral is tied in front at the symphysis by the superior pubic rami and the ischio-sacral at the pubic symphysis by the conjoint rami of the ischium and pubis. In determining the strength of the pelvis these arches are important and must be carefully considered in the conduction of tests on this region. On the basis of this information, a series of tests was designed to determine the strength of the sacrum, since through this joint both pairs of pelvic arches become confluent with the sacrum and for this reason alone, it constitutes one of the most significant articulations. The strength of this joint and type of fracture have already been described.

The strength of the lateral wall of acetabulum was also determined because it is directly related to the femoro-sacral arch. In spite of this relationship, however, no tests produced fractures destroying the integrity of the femoro-sacral arch though the force was delivered directly below its anterior extremity; it follows, therefore, that the sacro-iliac joint was not involved in the initial injury.

The remaining wall, the anterior wall, is important because this part of the pelvic ring is formed by the union of the bodies of the public bones from which rami, the superior and inferior, unit the illum and ischium of the lateral pelvic walls. It unites the weight bearing arches in front. From the description of fracture obtained when this anterior wall is subjected to stress to the limit of tolerance, it is at once apparent that a definite and well defined relationship exists. The secondary involvement of the sacro-iliac joint, fracture disjunction, by fractures of the symphysis or anterior wall carries with it the inference that damage to the rami, superior or inferior, damages the principal arches and spreads open the joint.

A remaining series of experiments was designed to correlate the strength the anterior and posterior wall give to the inherent strength of the entire pelvic ring. The posterior wall was first disrupted before tests were conducted on the anterior wall and conversely on another sample, the anterior wall was disrupted before tests were conducted on posterior wall. From the data presented herein, one may say generally that the posterior and anterior wall play equal part in the inherent strength of the entire pelvic ring.

Conclusions

Methods, strength, and type and location of fractures have been described for the various structures of the pelvis, inclusive of the acetabulum and femur. In the intact condition, fractures of the femurs

do not occur when this force is applied by cables to the interior surface of the neck of the femur, or when a force is applied to the base of the sacrum. Fractures of the lateral wall of the pelvis and acetabulum do not produce fractures or disjunction of the sacro-iliac joint. The strength of the pelvic ring is directly related to the strength of the anterior and posterior wall and as such, the strength of these walls must be known to correctly ascertain the inherent strength of the entire pelvic ring. Primary fractures of the pubic ramus-unilateral or bilateral, produce secondary fractures or disjunction of the sacro-iliac joint by disrupting the integrity of the counter-arch of the pelvis. The counter arch is, therefore, the important factor in maintaining the inherent strength of the pelvis for the reason that it ties anteriorly, the weight-bearing arches that are confluent with the sacrum.

SECTION VI

PHYSIOLOGICAL STUDIES

A. HIGH SPEED X-RAYS

Methods and Results

Previous data from this laboratory - Joffee (108) - shows that congestion of the lungs interpreted as congestive heart failure is the primary result of deceleration with abdominal safety belts. This symptom is also characteristic of non-penetrating chest injury. Since literature on the effects of abdominal safety belts is nil, a survey was made of both the clinical and experimental literature on non-penetrating chest injury in order to determine the evidence for production of cardiac failure. In line with our second objective, high speed X-ray studies were carried out with the assistance of the Allegheny Ballistic Laboratory, Cumberland, Maryland.

Since none of the theories gleaned from the available literature on nonpenetrating chest injury explained completely the mechanism which we surmised was responsible for cardiac failure, it seemed likely that information obtained by X-ray at the instant of deceleration might supply part of the answer to this question.

Dogs were anesthetized with nembutal and treated in accordance with the standards established by the American Medical Association.

Each dog was harnessed to an aluminum frame in such a manner as to permit animals to fall freely a distance of 5 1/2 feet in superior-inferior position. Two vertical "I" beams about 24" apart (the width of the frame) were used as guides for this frame. A 3/4" hemp rope was used as a tail line and was securely fastened around the dogs

abdomen. At the instant of deceleration, a force of 500 - 600 lbs. was delivered to the abdomen and the thorax by the action of the belt.

A Westinghouse high speed X-ray apparatus was synchronized in such a manner that serial X-ray photographs at the rate of 50 frames per second were taken at the instant of deceleration for approximately 1 1/2 seconds. The actual size of the individual frames was 2 $1/2 \times 8$ inches. Several difficulties are obvious in this type of experiment.

- 1. The X-ray plate was not large enough to show a clear cut outline of the entire heart. It was possible, however, to observe changes in maximum transverse diameter, maximum right (T_R) and maximum left (T_L) projections, as well as changes in surface area of cardiac silhouette.
- 2. It was not always possible to direct the fall of the dogs to the set position of the X-ray machine and to the degree that the heart itself was always visualized.
- 3. Because of the extremely fast speed of the machine and the thickness of the thoracic cage of the dog, many of the individual frames in the roll were under-exposed and, consequently, no cardiac silhouette could be observed.

Control and experimental runs were made in both the anterior-posterior and lateral positions. Protocols for each of the seven dogs, accompany this dissertation, together with statistical summary of results.

Results were sufficiently striking in Roll No. 1 so that separate measurements were made on a total of 63 frames.

Figure 13 and 14 shows the type of X-ray shadow taken at the instant of deceleration. Figure 15 shows the type of measurements taken from the individual frames of the entire roll (contains 63 frames).

Maximum transverse - x Maximum right and left projections - T_R and T_L respectively Area of cardiac silhouette given by equation:

 $A \sim \pi/4$ L B Where A = area

L = long diameter

B = broad diameter.

These same measurements are used in the clinic as valuable diagnostic aids in determining size of the cardiac shadow.

Individual measurements are recorded in Table I. The change in values, indicated in the same table are plotted as ordinates with time as abscisa.

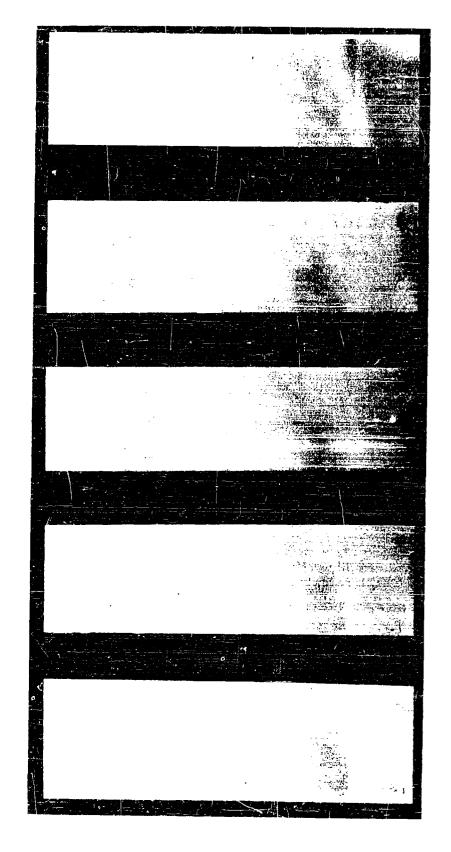


Figure 13. Heart shedows by high speed X-ray at instant of deceleration.

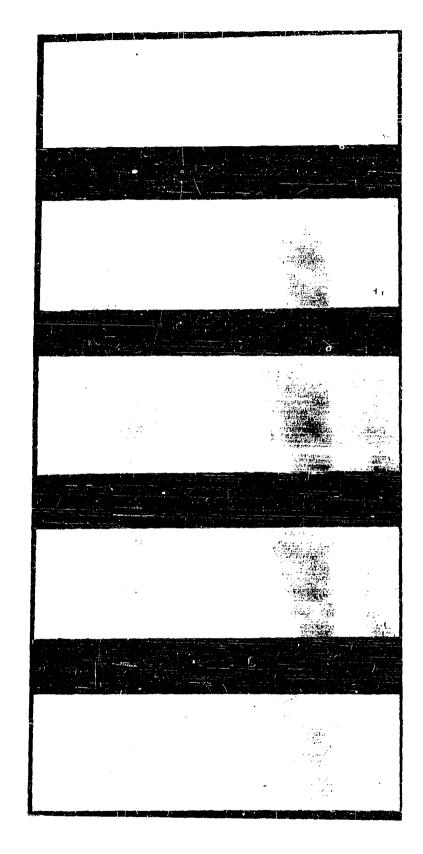


Figure 14. Heart shadows by high speed X-ray at instant of deceleration (continued).

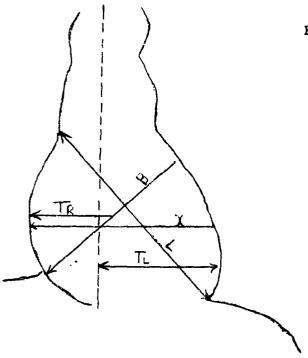


Figure 15. Heart Size Measurements

Legend:

Transverse Diameter = Tr + Tl (sum of maximum projections to right and left heart borders from midline)

Transverse Diameter = X (maximum projection at the level indicated)

Long Diameter = L (extends from junction of cardiac silhouette and vascular pedicle on right to apex on left)

Broad Diameter = B (greatest diameter of cardiac shadow perpendicular to long diameter)

Calculated area from Long and Broad Diameters:

TABLE I HEART SIZE MEASUREMENTS

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TABLE I (Continued)

ilm No.	Maximum Transverse Diameter	Change	Interpolated	lated 'B'	ят ү/и	Change in π/4 L B	F.	$\mathbf{T_L}$	$T_{ m R} + T_{ m L}$	$\begin{array}{c} \texttt{Change} & \texttt{In} \\ \texttt{T}_{R} + \texttt{T}_{L} \end{array}$
8	71	<i>(</i> —	76	75	51.2	12.2	8	50	91	9
81	71	[95	75	55.9	13.9	%	50	92	9
88	72	ထ	8	92	27.6	12.6	25	20	75	7
83	70	9	16	17	55.1	10.1	8	64	75	ľ
ð	70	9	&	73	51.0	0.9	8	64	75	. در
85	70	છ	92	73	52.9	7.9	25	84	73	ĸ

Discussion

An inspection of the protocols shows, and it has already been mentioned, that complete data could be obtained from only one roll of the total of 14 rolls. It must at the same time be clearly pointed out that this was due in the majority of cases to under-exposed film caused by the rapid speed at which the X-ray machine was operated. It must also be pointed out that Figures 13 and 14 are reduced in size from the originals and the heart shadows, therefore, are not as clearly defined as in the original.

Four of the rolls showed evidence of transverse enlargement, but unfortunately, these changes could not be analyzed in the same manner and to the same degree as in roll No. 1. Therefore, it is desirable to fortify our argument concerning the production of cardiac damage with many measurements on roll No. 1. A total of 189 separate measurements was made on this roll and the results plotted on separate graphs. Any one graph by itself might be considered a matter of chance; however, the element of chance is reduced if separate and distinct measurements in graphs follow the same type of curves.

The data plotted as graphs in Figures 16, 17, 18 was taken from Table 1. The oscillation as shown by the graphs suggests the dogs bounced several times before coming to a complete standstill.

Protocols for the five dogs subjected to this experimental procedure are found in Table II.

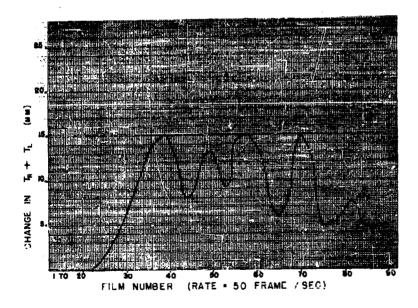


Figure 16. Graph (howing change in $T_R + T_L$.

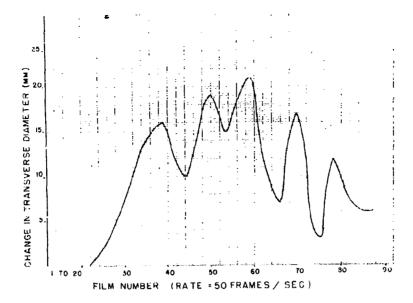


Figure 17. Graph showing change in transverse diameter.

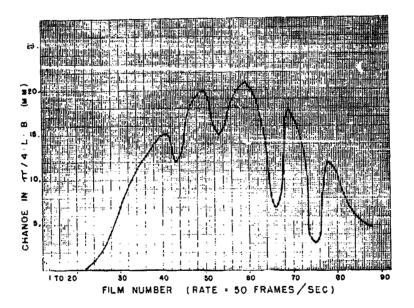


Figure 18. Graph showing change in $\pi/4$ L·B.

Inspection and superimposing these three graphs show that peaks and troughs occur in most instances at precisely the same interval of time. We must also make clear that plotting of data from Table 1 does not give the smooth curve indicated in the three graphs. It showed

TABLE 2

SUMMARY OF HIGH SPEED X-RAY FILMS

Dec. 35.		Heart Changes	Lung	
LON BOLL			Neuelfre	
Sex Fermle Weight, 24 lbs.	Roll No. 1 Drop - 5ft. 6 inch Position - anterior posterior	Results of data from this experiment is graphed in figures 16, 17, 18 Increase in the prominence of the artery segment noted.	No. 31 obliteration of costo-phrenic angle. No. 33 two-thirds of lung field obliterated. No. 34 left inferior half of lung obliter-ated.	
	Rell No. 2 Drop - 5 ft. 6 inch Position - lateral	Maximum change noted from 7.6 cm to 8.5 cm.	Clear throughout Light exposure	
Dog No. 2				
Sex Male Weight. 15 lbs.	Roll No. 3 Control Position - anterior posterior	Meximum change noted 0.5 cm	Clear throughout	
	Roll No. 4 Control Position - enterior posterior	No evidence of change	Clear throughout	•
	Roll No. 5 Drop - 5 ft. 6 inch Position - anterior	Maximum change noted. This is maximum change noted, but not enough data to plot as in Dog No. 1.	No. 4 clear Light exposure No. 5 clouding along cardiac border	posure

	Heart Changes	Lung	Remerks
Dog No. 2 (cont.) Roll No. 6 Drop - 5 ft. 6 inch Position - lateral	No. 3 - 8 cm No. 4 - 7 cm No. 85 - 6.5 cm	No. 3 lung field clear posteriorly but the inferior region is cloudy throughout.	Changes appear in both anterior cardiac space and retro-cardiac space.
Dog No. 3 Sex Male Roll No. 7 Weight. 22., 1bs.Control Position - anterior posterior	No changes noted	Clear throughout.	1
Roll No. 8 Control Position - anterior posterior	The heart could not be adequately seen because of light . exposure		
Holl No. 9 Drop - 5 ft. 6 inch Position - Lateral	Heart was obscured by the dog's elbows		
Roll Mo. 10 Drop - 5 ft. 6 inch Position - anterlor posterior	Mo. 7 transverse dia. 8 cm No. 25 transverse dia. 8.5 cm No. 30 transverse dia. 9.5 cm Maxiwam change noted - 1.5 cm	No evidence of change	

		House to the		
OF No. 4		near coanges	Lung	Remarks
ب	Roll No. 11 Control Position - anterior posterior	No evidence of change in transverse diameter	Clear throughout	
	Roll No. 12 Control Position - lateral	No evidence of change	Clear throughout	
	Roll No. 13 Drop - 5 ft. 6 inch Position - lateral			Exposure of this film too light to permit measurements.
	Roll No. 14 Drop - 5 ft. 6 inch Position - anterior rosterior	Minimal suggration in this roll of cardiac enlargement in the area of the outflow tract on the left	Clear	
g No. 5				
Sex Female Weight . 13.5 lbs.	Roll No. 15 Control Position - anterior posterior	No evidence of change	Clear throughout	
	Roll No. 16 Control Position - lateral	No evidence of change	Clear throughout	
	Roll Mo. 17 Drop - 5 ft. 6 inch Position - lateral	No definite evidence No. 9 of change in trans-field verse diameter, but a prominence of the pul-monary artery segment is noted	No. 9 right central field cloudy noted	

		Heart Changes	Lung	Remarks
Dog No. 5 (Cont.)				
	Roll No. 18 Drop - 5 ft. 6 inch Position	Roll too light to permit measurements		
Dog No. 6				
Sex Male Weight 21.5 lbs.	Roll No. 19 Control Position - anterior posterior	No evidence of change	Clear throughout	
	Roll No. 20 Control Position - lateral	No evidence of change	Clear throughout	
	Roll No. 21			Belt broke; therefore no measure-ments were taken on this roll
	Roll No. 22			Same as Roll No. 21
	Roll No. 23 Drop - 5 ft. 6 inch Position - lateral	Questionable change appearing in this experiment amounting to approximately 1.5 cm	Clear throughout	
	Roll No. 24 Drop - 5 ft. 6 inch Position - enterior	Unable to evaluate heart changes because of light exposure	Lung bases clear	

	بيابيه والمرابع المرابع المراب			
		Heart Changes	Lang	Remerks
Dog No. 7				
Sex . Female Weight 11.5 lbs	Roll No. 25 Control	No absolute evidence of change	Clear throughous	
,	Position - anterior posterior			
	Roll No. 26 Control	No evidence of change	Clear throughout	
	Position - lateral			
	Roll No. 27	Possible 1 cm change be- Lungs appear first	Lungs appear first	
	Drop - 5 ft. 6 inch Position - lateral	tween film No. 10 and No. 30	cloudy posteriorly;	
		•	phrenic angle remains	
		Poor positioning of	Right lower lobe	
	Drop - 5 ft. 6 inch	animal made 1t	gradually becomes	
	Position - anterior	impossible to make	cloudy and remains	
	posterior	cardlac measurements	that way	

instead, an irregular force with many small vibrations. This question immediately presented itself: "How should this data be treated to indicate the phenomenon most clearly?" Several factors were considered before these data were plotted:

- 1. Precise measurements could only be made with an accuracy of ± 2 mm, therefore, differences of 2 mm were not considered to be significant.
- 2. Examination of subsequent accelerometer readings under the same conditions as these experiments show damping effect of biological tissue consisting of several peaks rather than one bell shaped curve. It it, therefore, justifiable to consider changes in transverse diameter and area of the cardiac silhouette in terms of damping with several peaks.
- 3. These data could be treated as moving averages which would give a smooth drive to some extent. However, since these data are presented as qualitative mather than quantitative measurements, our treatment of smoothing the curves without recourse to moving averages is justified and the theory for production of cardiac damage as a consequence of these data is believed valid.

A brief description of the mechanisms postulated by investigators in the field of nonpenetrating chest injury will first be given because it is entirely possible that to some extent these mechanisms also play a part in the production of injury. Five mechanisms have been suggested:

- 1. Work at Randolph Field (101) suggests that the vagus is important in the mediation of conduction disturbances and ectopic contractions. In their group of three dogs, conduction disturbances were found less frequently in vagotimized animals and more frequently than in the normal animal in the group of animals that had been given physostigmine.
- 2. The forcing of the blood into the ventricle causing an increase in intracardial pressure has been suggested, and in the opinion of the authors appears to be a likely cause of damage. In support of this, the work of Beck and Bright (10) is cited. By applying pressure to the legs and abdomen and forcing the blood back into the ventricle

very rapidly caused rupture of the valves and of the myocardium and overdistension of the heart. The degree of damage was directly related to the ability of the heart to accomodate to the increased blood supply.

- 3. The phase of the cardiac cycle has been mentioned as a means of inciting damage since the relaxed heart showed less damage than the distended heart. A Randolph Field report (102), however, showed that there was no relationship between the degree of electrocardiographic change and the phase of the cardiac cycle at which impact occurred.
- 4. Sigler (24) (25) has stated that trauma of the heart is frequently overlooked because of the feeling that the chest wall and the cushion effect of the lungs prevents a trauma. However, the fact that the heart has a rather loose attachment makes it vulnerable to practically any force. Joffee (108) has shown that a force delivered, via the chondrocostal arch, shows no damping effect, and the force delivered to the arch is transmitted as such to the heart.
- 5. Several investigators support the idea that a positive pressure wave is set up in the great vessels and is transmitted to the heart and responsible for damage. Potain, in 1894, proposed such an idea which he later supported with experimental evidence showing heart rupture with pressure differentials of 896 mm Hg.

Our experiments suggest that the mechanism by which cardiac damage is produced in rapid deceleration involves the action of the belt in suddenly blocking blood flow in the abdessen, both in the vena cava and the abdominal aorta. Since the flow of blood from the upper region of the body is toward the heart, the effect produced is similar to that of a closed liquid system subjected to a force from below and above. Therefore, the heart is squeezed between these two forces and stretched in the transverse direction. This phenomena is analogous to the deformation of a rubber belloon filled with water which is suddenly and forcibly deformed by forces acting from below and above. In these experiments, the changes noted either diminished or completely disappeared within a fraction of a second after the force was applied. Even so, it is possible that this force might produce cardiac pathology by momentarily stretching the muscle beyond its limit of distensibility. Furthermore, it is likely that higher forces of deceleration might result in permanent deformation of the myocardium.

Summary of High Speed X-ray Studies of Dog Thorax After Rapid Deceleration

Dogs were decelerated with 3/4 inch hemp rope from a free fall of 5 1/2 feet, with a force between 500 and 600 lbs. All dogs lived and showed no gross pathology at autopsy.

At no time could any evidence of eventration or nerniation of the hemidiaphragms be identified.

No evidence of fracture of the thoracic cage was noted.

Only four rolls demonstrated possible diffuse cardiac enlargement. Of the four that demonstrated questionable enlargement of the pulmonary artery segment or outflow tract, only two showed cardiac enlargement as well (No. 3 and No. 1).

Of the three that demonstrated cloudiness in the lung fields, only one was of more than the costo-phrenic angles.

The most striking changes of all occurred in No. 1 where there was diffuse infiltration in the lung fields bilaterally, associated with cardiac enlargement and prominence of the pulmonary artery segment. All of the changes disappeared before the roll was completed.

The findings that of the 23 visualized hemidiaphragms, 13 changes in location suggests that the animals "bounced" one or more times at the completion of their drop.

STATISTICAL SUMMARY OF FILMS

Number of rolls	28			
AP	7	Light films	1	8
Laterals	8	Light films	5	13
Obliques	7	•	•	7
				28

Range of exposures/roll - 49 - 88

CARDIAC CONTOUR

Unchanged	11	(This Includes 10 Controls)
Inadequately seen	10	•
Incr. diameter	4	
? inch pulmonary	<u>L</u>	(of which only two were the same as
artery segment		(increase in total diameter

LUNGS

Clear	15
Clouded, but clear	1
Clouded but did not clear	3
Not clearly seen	()

DECELERATION EXPERIMENTS

Methods and Results

This phase of the investigation was undertaken to fulfill the third objective: "To measure the tolerance of dogs to rapid deceleration and to provide information which would contributed to the solution of the controversy concerning the possible danger resulting from the use of abdominal safety belts in rapid deceleration". Considerable data was accumulated in this laboratory from the period 1946 to 1948 by Joffee (108). Several difficulties were encountered during the phase of the investigation concerning accurate measurements of the time and peak periods of deceleration. In the words of the investigator: "As will be brought out later, our results on living animals indicate beyond doubt that decelerative forces, when applied by means of the safety belt or harness at the lower margin of the ribs, produce cardiac injury so characteristic of heart trauma due to nonpenetrating chest wounds or any origin, and that a knowledge of the force level at which this injury might be manifest would be of great value".

We have attempted then, to provide the additional piece of information lacking in that investigation, i.e., to provide accelerometer measurements of the lethal dose of deceleration as determined by an accelerometer mounted at the region of the fifth rib.

In the preceding section concerning the effects of abdominal deceleration as determined by high speed X-rays, we described the manner in which the forced is delivered to the lower region of the ribs by these safety belts when the dog is oriented in the superior-inferior position. This same method was used in this phase of investigation.

By suturing the accelerometer to the fifth rib, a rigid mounting was obtained, and under such conditions, deceleration readings had no distortion which might otherwise have been caused by the acceleration of the mass of the transducer itself.

To date, four dogs have been subjected to decelerative forces of short duration. Dats on the fourth dog is not complete, but the

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available data will, nevertheless, be given and final, unequivocal conclusions withheld until additional data is forthcoming.

Based upon gross autopsy findings, Joffee (108) reported seven out of seven dogs subjected to horizontal deceleration of unknown magnitude died of cardiac dilatation. We have attempted in this phase of the investigation to replace the subjective assessment of cardiac dilatation with objective measurements of the area of the cardiac silhouette by planimeter on X-ray shadows taken before and after deceleration, and to further substantiate these findings with histological examination of section of heart tissue. Since evidence exists, clinical and experimental, that some damage is sustained by the liver and kidneys, histological examinations were made also of these two organs.

Electrocardiographic changes for each of the first three dogs have been summarized as to rhythm, P-R, QRS, QRST intervals, heart rate, and individual measurements in amplitude of P, Q, R, S, T waves and the level of the ST segment in leads I, II, III.

It has been mentioned previously that the literature shows cardiac trauma is associated in humans with nonpenetrating injury to the chest. Joffe, in this laboratory, showed that cardiac trauma produced experimentally in animals with abdominal safety belts shows results similar to those of nonpenetrating chest injury. Both conditions produce electrocardiographic changes involving the ST segment and the T wave.

On the basis of this information, our evidence for the production of cardiac trauma is based on the inversion of the T wave with simultaneous elevation of the ST segment. Many other bizzare changes occur such as enlargement of the Q wave and changes in contour of the QRS complex, but unfortunately, these changes cannot be accurately described and cannot be correlated with any specific heart damage.

Dog No. 1 (Scottie) was subjected to three separate falls from a height of 5 1/2 feet with a force of approximately 20 g's acting for 0.1 seconds. This was done to determine whether or not there were any accumulative affects from repeated exposure to rapid decelerations. Separate electrocardiograms were taken before and after each run and are mounted in Figures 19 and 20. Protocols summarizing electrocardiographic changes are also presented. Examination will show that no significant changes occurred except 10 minutes after the deceleration of the first run. Slight inversion of the T wave was noted with simultaneous elevated takeoff of the ST segment. It is, however, doubtful that these slight changes can be interpreted as definite evidence of cardiac damage. No X-rays are available on this dog.

Second and third drop were made from the same height about a week apart. Serial electrocardiograms following these two drops show no evidence of damage and the inference is that animals tolerate this magnitude and duration especially well and that little accumulative effects are produced from repeated sublethal doses of rapid deceleration. This dog was sacrificed about 3 weeks following third drop.

Dog No. 2 (Hound) was dropped in the same manner as indicated in the preceding experiment, sustaining a force of 56.7 g's for a period of 0.06 seconds at the level of the fifth rib.

The most significant changes in the electrocardiogram occurred five minutes after the drop. In this series, the T wave appears in lead I, whereas the control showed no wave. The T wave is inverted and is accompanied by a slightly higher takeoff of the ST segment. These changes, according to the literature (1) (24) (30) (34) (35), indicate that some cardiac damage has been produced with a free fall from a height of 12 feet with the magnitude and duration of force indicated. The following series of electrocardiograms in Figures 21, 22, 23 and 24, and the accompanying summaries shows these changes to become less noticable as time goes on. It is doubtful that any permanent damage was produced.

Ristological examination of heart sections showed only isclated small hemorrhages in the myocardium, no degeneration of the parenchymatous myocardium was noted. Small subcapsular hemorrhages of the liver were noted. No changes in renal tissue was noted. None of these changes is believed to be severe enough to cause discomfort or death to the animal.

Serial X-rays of this animal showed changes in the area of the cardiac silhouette amounting to approximately 5 sq cm. This dilatation practically disappears in about three weeks. This dog was sacrificed about three weeks following last drop.

Dog No. 3 (Brownie) was dropped from a height of 12 1/2 feet. Accelerometer record is not available, but on the basis of subsequent experiments using the same apparatus, the estimated force is 50 g's acting for 0.05 seconds.

Serial electrocardiograms (Figures 25, 26, 27, and 28) were taken for a period of three weeks following the deceleration at which time the animal was sacrificed and histological sections made of heart, liver and kidney.

The most significant electrocardiographic changes were noted in which a definite sinus arrhythmia with simultaneous changes involving the ST segment persisted until the time the dog was sacrificed.

Histological examination showed no damage to the kidney or any degeneration of the myocardium. Small areas of hemorrhage were noted in the myocardium and in the subcapsular area of the liver. None of these changes are believed of sufficient intensity to cause distress or discomfort to the animal. Measurement of the cardiac silhouette revealed no significant changes.

A force of 55 g's was applied to the thorax of Dog No. 4 (Lady). As has been pointed out, complete data is not available on this dog. Control electrocardiogram showed inversion of the T wave in all leads, but it must also be emphasized that the inversion became deeper and the ST segment elevated following the drop. This dog died 10 days later, but it cannot be determined whether death in this case was caused by or morely hastened by the effects of the deceleration. At autopsy, parasites were found to have invaded the intestinal tract and undoubtedly was responsible for the general emeciation of the dog prior to death.

Changes in the cardiac silhouette were minimal. Significant changes were noted in Dog No. 4 in the histological examination of the heart, liver, and kidney. Sections of the heart showed the coronary tree to be engarged with blood with small hemorrhages appearing throughout the myocardium. According to veterinary pathologist none of the three parasites (Dinofilaria, Sarcosporidia, Toxoplasma) which are known to invade heart tissue in the dog could be responsible for the lesions found in this dog. None of these parasites, according to a veterinary pathologist are known to cause hemorrhages as was noted in these sections. Larger vessels of the heart appeared normal. The hemorrhage areas were noted in the arterioles and venules of the myocardium.

Some hemorrhage was noted in the renal tissue and there was general congestion of the vessels of the cortex involving the interlobar vessels.

Large hemorrhage areas were noted in the liver at the periphery of the inferior border and extending into the septa of the larger vessels. Hepatic tissue appeared normal, but liver sinuses were engorged in some sections and ruptured in others. Small hemorrhage areas were also noted along the chief portal vessels.

Conclusions

As has already been mentioned, final conclusions must be waived on this phase of the investigation until additional data is forthcoming. To date, it has not been possible to deliver enough deceleration from freefall conditions to produce anything comparable to the cardiac damage under the conditions of Joffse's (108) experiments.

Nevertheless, one may, at this point, hazard a guess as to the limit of tolerance of dogs to decelerative forces applied at the lower region of the ribs. Since we have produced some minor damage to the heart at the level of g's indicated (56 g's) in these experiments, the lethal dose of deceleration might possibly be reached at approximately 100 g's acting for 0.1 seconds.

Enough evidence has been reviewed to at least suggest some of the possible dangers resulting from use of the abdominal type safety belt. Though the heart, by virtue of its position, seems particularly vulnerable to violence, the liver and kidney may also be included as areas in which damage to viscera might be produced.

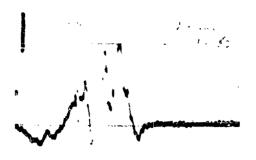
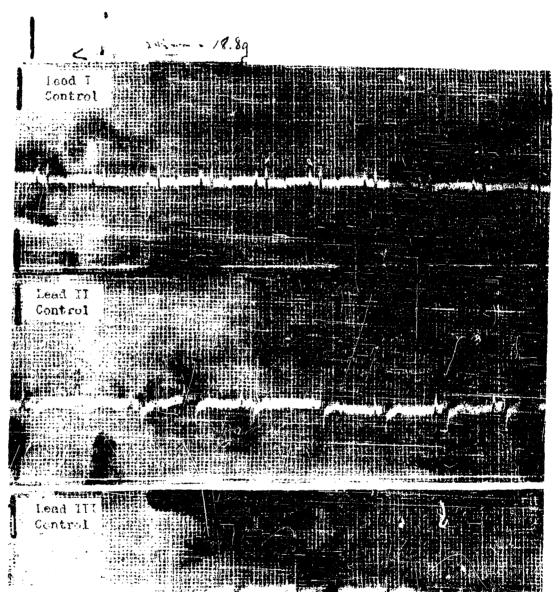


Figure 19. Electrocardiogram of Dog. No. 1 (control).



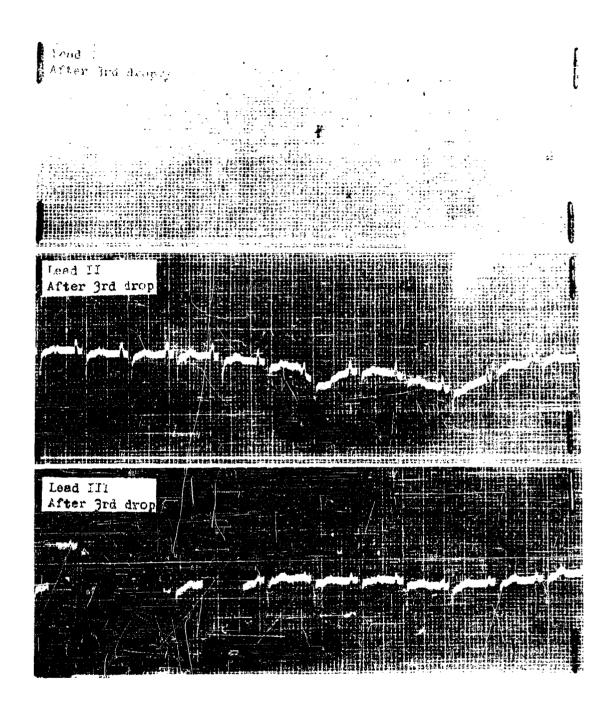


Figure 20. Electrocardiogram of Dog No. 1 after third drop.

i	bs. EKG Control					Lead 111	+.15	slightly elevated	1.3	5.	Low takeoff then blends with	T wave Inverted to diphasic
Loi mb+	"crear cy los.							ģ			s with T wave	
Deceleration None						Ilead II	+.15	silgatly elevated	۲۰۶	ŗ	Low takeoff then blends with T wave	Inverted to diphasic
0	115/min.	Regular	.12 sec.	.04 sec.	.28 sec.	Lead I	+ •3 uv - •15	, w	. 0		Ispelectric	Inverted .05 mv.
Dog Scottie	Heart Rate	Rhythm	P-R Interval	QRS Interval	QRST Interval	l P	· ·	R	æ		ST	; ; ;
LIATS		l	0									

	. EKG 10 min. after drop						Lead III	A 110 PE		ντη ζ ντη ζ.μ.	Isgelectric, blends with	I wave	d as cardiac
Deceleration 18.5 a's .10 sectletant	Comerague 27 Ibs.					TT 6001	+.20 mv	0	1.9 av	νω 4°.	Isoelectric, blends with T wave	Inverted to diphasic	Some T wave changes appear, but cannot definitely be interpreted as cardiac
	88/min.	Regular	.10 sec.	.05 sec.	.28 sec.	Lead I	+ .25 шу	0	•6 av	•05 av	Slightly higher takeoff than control	Inverted to diphasic	Some T wave changes appear, damage,
Dog Scottie	Beart Rate	Rhythm	P-R Interval	QRS Interval	QKST Interval	'	Ω.,	· · · · · · · · · · · · · · · · · · ·		; ; ;	ST	E	

	Dog Scottle	e	Deceleration None	Weight 25 lbs.	EKG Control for 2nd drop
	Heart Rate	94/min.			
	Rhythm	Regular			
-	P-R Interval	.11 sec.			
	QRS Interval	.05 sec.			
	QRST Interval	.28 sec			
	I	Lead I	II pael	I	Lead III
	Ъ+	+ .15 шv	+ .2 @v	2i	+ .l (several diaphasic waves)
63	•	.l mv	٥		0
	я	.5 mv	1.5 ш	2	1.6 wv
		0	ለመ ተ"	A	vm 9.

No appreciable change from the original

Inverted to diphasic

Low takeoff blends with T wave

Low takeoff blends with T wave

ST.... Isolectric

T.... Inverted

Inverted

EKG 10 min. after 2nd dren						Lead III	+ .20 mv	0	1.5 mv	vm ?•	Slightly depressed take.	Inverted
Deceleration 21.4 g's .13 sec. Weight 25 lbs.						I.ead II	+ .25 mv (notched)	0	1.5 mv	.3 mv	Depressed takeoff blends with T wave	Inverted to diphasic
	125/win.	Regular	.10 sec.	.05 sec.	.24 sec.	Lead I	+ .10 av	.05 по	ΛEE ty*	0	Isœlectric	Inverted
Dog Scottie	Heart Rate	Rhythm	F-R Interval	QRS Interval	QRST Interval	·	6		ж	 	ST	E-1

No appreciable change

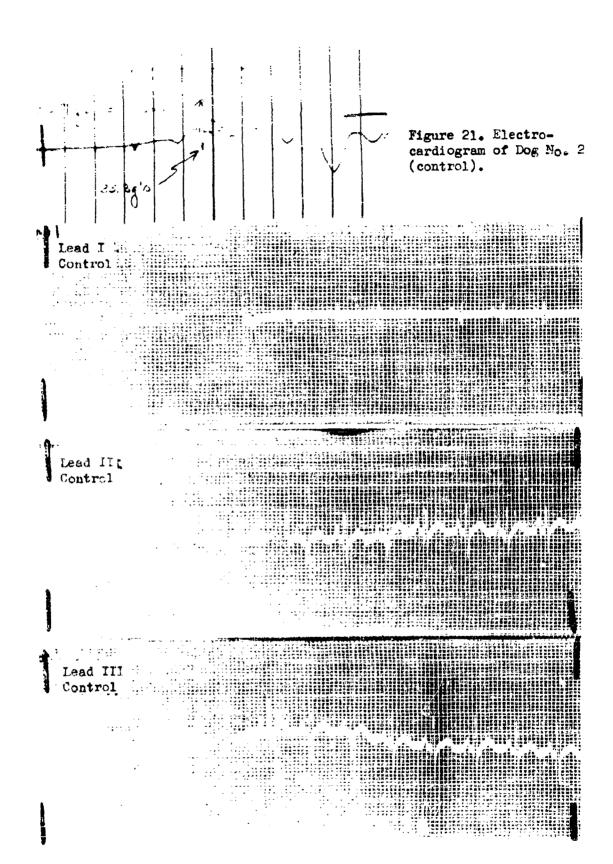
EKG Control for 3rd drop						Lead III	v. 15 av	0	1.3 m	·5 mv	Depressed takeoff blends with T wave	Inverted (diphasic)
None Weight 25 lbs.						Lead II	+ .25 (some notching) av	0	1.7 mv	væ ₹.	Slightly higher takeoff than orignal	Inverted (diphasic)
Deceleration	115/min.	Regular	.12 Bec.	ീ ഉട	.28 sec.	Lead 1	+ .15 uv	0	AM 17°	•05 ш/	Isoelectric	Inverted (diphasic)
Dog Scottle	Heart Rate	Rhythm	P-R Interval	QRS Interval	QRST Interval	1	£.	G.	æ	: : : : : : : :		T

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No appreciable change from the original

EXG 4 min, after 3rd decr	do in the case of					Iead III	+ .15 av	0	1.4 grv	vm ₹.	Slightly higher takeoif than orignal	Inverted to diphasic
Deceleration approx. 15 g's Weight 25 lbs.				ome splintering of this complex)		II Pad II	+ .25 uv	0	1.6 mv	AM 17.	c Slightly higher takeoff then orignel	Inverted to diplasic Inverted to diphasic
	136/ain.	Regular	.09 sec.	.05 sec. (some	.28 sec.	I paər	+ .15 mv	0	.6 av	.1 mv	Is c electric	Inverted t
Dog Scottle	Heart Rate	Rhythm	F-R Interval	QRS Interval	QRST Interval	1	<u>.</u>	9	24	Ω.	e.	H
WA	DC T	R 54	~518					6	6			

No appreciable change



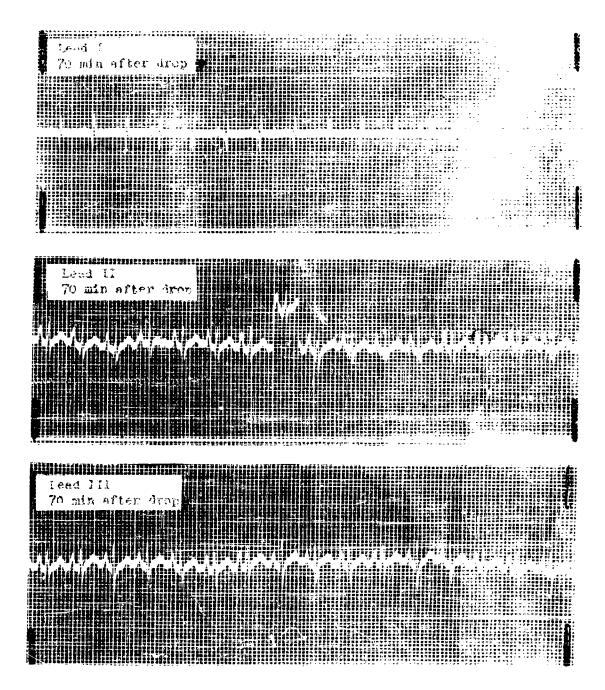


Figure 22. Electrocardiogram of Dog. No. 2 70 minutes after drop.

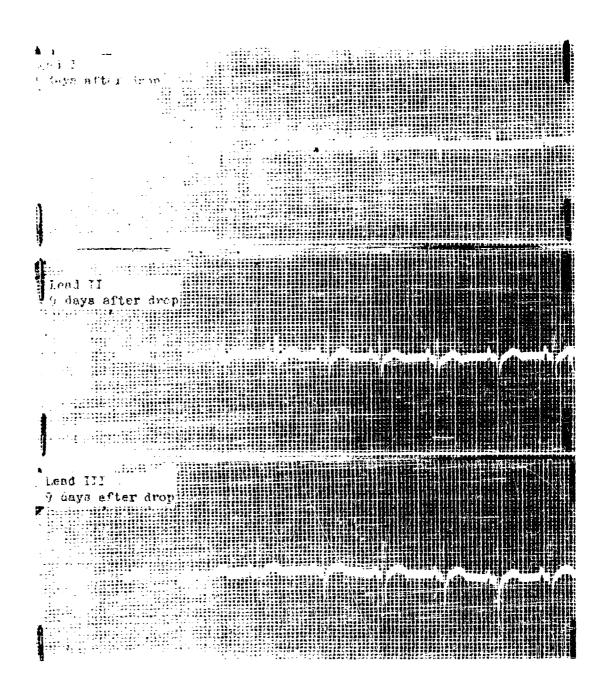


Figure 23. Electrocardiogram of Dog. No. 2 nine days after drop.

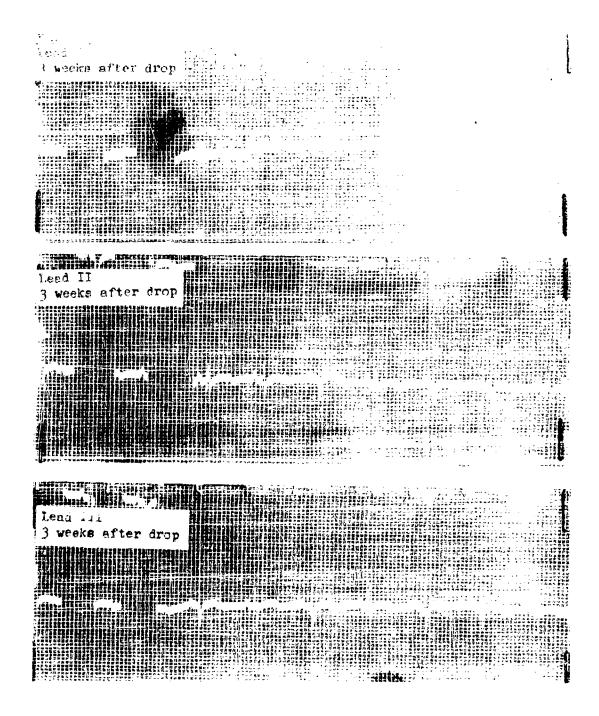


Figure 24. Electrocardiogram of Dog. No. 2 three weeks after drop.

WAT	Dog Hound		Deceleration	Mone	Weight 22 lbs.	EKG	Control
ורי תנוס	Heart Rate	150/min.					
Sh _	Rhytbm	Reguler					
218	P-R Interval	.10 sec.					
	QRS Interval	.03 sec.					
	QRST laterval	.28 sec.					
		Lead I		I.ead II	II		Lead III
	Δ,	* •05 BW		+ •3 mv	DIV.		+ .2 av
	.	.2 mv		.15	.15 av		.15 av
	DC	.25 uv		ADD E.	AU		1.0 w
	ω •	0		.5 arv	er.		.6 av
	SH	Isoelectric	77	Low takeoff by wave	Low takeoff blends with T wave	Lor	Low takeoif blends w T wave
	E-1	No T wave present		Elevated T wave	Ve	E14	Elevated T wave

EKG 5 min. after drop						Lead III	+ .15 mv	.1 arv	Va 8.	v™ 54.	Low takeoff blends with T wave
Deceleration 56.7 g's .05 sec. Weight 22 lbs.						Lead II	+ •2 mv	.1 uv	ν ω 8.	Am 1/°	: Low takeoff blends with T wave
	158/win.	Regular	.09 sec.	.03 sec.	,20 sec.	Lead I	vas 20. +	0	AM †	.1 mv	Isœlectric
Dog Hound	Heart Rate	Rhythm	P-R Interval	QRS Interval	QRST Interval	1	P +05 EW	Ç	8	S	ST
WAD	C TR	54-2	218					72	2		

The most significant changes take place in lead I with inverted I waves. None were present in Lead I of the control.

Slightly higher takeoff

T..... Inverted

Elevated

Dog Hound		Deceleration same	Weight 22 lbs.	ENG 25 min. after drop
Heart Rate	175/win.			
Rhythm	Regular			
P-R Interval	.09 sec.			
QRS Interval	.O4 to .O5 sec.	sec. slight increase over control	control	
QRST Interval	.22 sec.			
•	Lead I	II Peal	I II	Lead III
д.	+ •1 av	+	+ •3 tav	+ •3 mv
Ò	.25 av	7	.15 wv	.l av
67 	om 4°	-1	't m t.	.5 mv
S	0	***	•3 fev	ъ. 5 пт
ST	[soelectric	Tends to bec	Tends to become isoelectric	Same
	Inverted	Tends to be	Tends to become inerted	Tends to become inverted

T wave remain inverted but are less prominent than in control QRS interval becomes splintered

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EKG 70 min. after drop						Lead III	+.25 uv	.10 av	vв 3.	•5 mv	
Weight 22 lbs.						Lead II	+ .25 uv	.15 EV	.6 mv	.e mv	
Deceleration same											
	180/win.	Regular	.10 sec.	.04 sec.	.20 sec.	Lead I	+ .03 uv	ΛΠ tr	,5 tav	0	
Dog Hound	Heart Rate	Rhythm	P-R Interval	ORS Interval	QRST Interval	ľ	ρ4	0	В.	M	
WAD	C TR	54-2	218					74	,		

;

Changes less prominent than in the EKG 5 minutes after drop

Isoelectric takeoff blends with T wave

Isoelectric takeoff blends with

T wave

ST.... Slightly slurred

Tends to become inverted

Inverted when 1t

Ţ....

appears

Tends to become inverted

SKG 1 Day						Lead III	+.05 av	.15 w	л <u>я</u> 6•	.1.5 uv	Depressed takeoff blends with T wave	Inverted and notched
Deceleration Same Weight 22 lbs.						Lead II	+.25 wv	.20 mv	νω ή·	.15 arv	Depressed takeoff blends with T wave	Inverted and notched
Dece	100/min.	Regular	.10 sec.	.Ot sec.	.20 sec.	Lead I	+.05 mv	.25 av	.2 IIV	.25 mv	Slightly elevated then isoelectric	Inverted
Dog Hound	Heart Rate	Rhythm	P-R Interval	QRS Interval	ORST Interval	•	Ω	Ġ	æ		SH	E-1
WAD	C TR	54-2	218					75				

75

T waves appear inverted and notched

P waves appear notched

• EKG 5 days						Lead III	+.15 mv	.15 mv	1.0 av	AR 9.	Low takeoff blends with T ware	Elevated T wave
Deceleration Same Weight 22 lbs.						Lead II	+-2 配	•15 av	AE 2.*	√5 wv	Low takeoff blends with T wave	Elevated T wave
	97/win.	Regular	10 sec.	ob sec.	24 sec.	Lead I	P + . 05 arv	4 mv	· . h mv	0.	 Slightly elevated takeoff 	. Slightly inverted
Dog	Heart Rate	Rhythm	P-R Interval	QRS Interval	QRST Interval		Ω		per	Ω.	ST	13.00

Rhythm slower associated with a slight inversion of T wave in lead I.

ЕКС 3 меекв						Lead III	+ .15 mv	.l urv	•5 mv	√m 5.	Low takeoff blends with T wave	Diphasic
tion same Weight 22 lbs.	-					Lead II	4 .2 av	.15 mv	AE ¿.	.35 шv	Low takeoff blends with T wave	Dipbasis
Dog Hound Deceleration	Heart Rate 107/min.	Rhythm Regular	P-R Interval .10 sec.	QRS Interval .05 sec.	QRST Interval .20 sec.	Lead I	P	Q1 mv	R 3 mv	80	ST Slightly elevated	T Inverted

Except for the slightly elevated takeoff of the ST segment in lead land the T waves of the Lead II and III, this EKG looks similar to the control.

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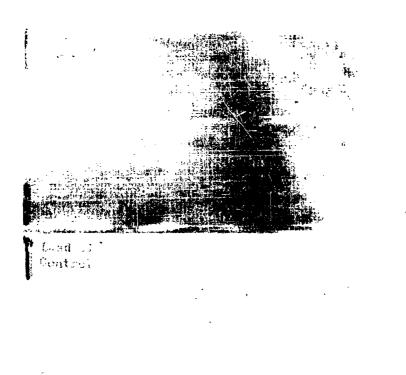


Figure 25. Electrocardiogram of Dog No. 3 (control)

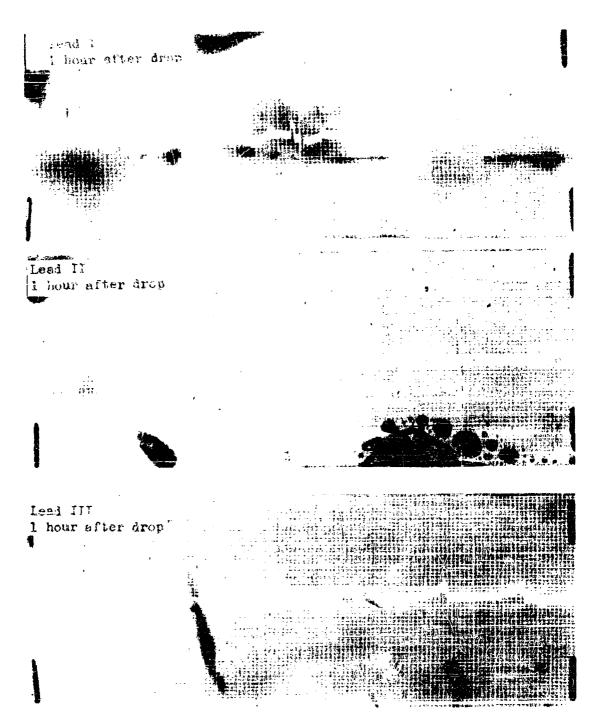


Figure 26. Electrocardiogram of Dog No. 3 one hour after drop.

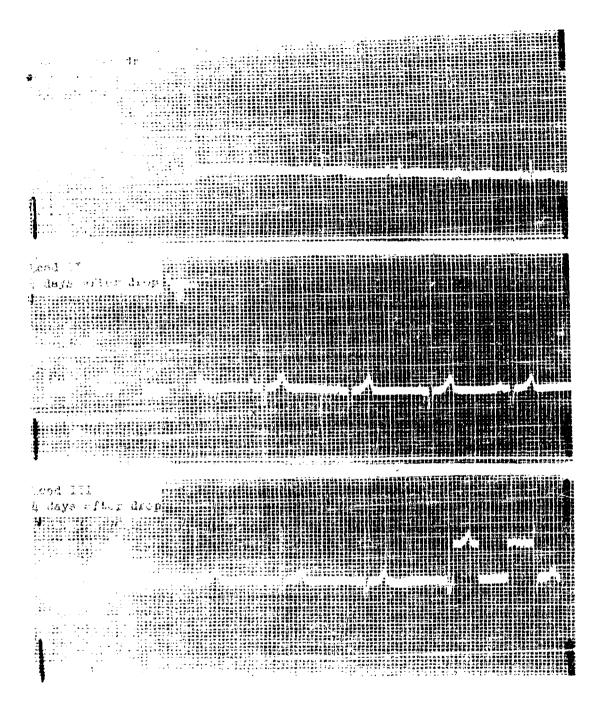


Figure 27. Electrocardiogram of Dog No. 3 four days after drop.

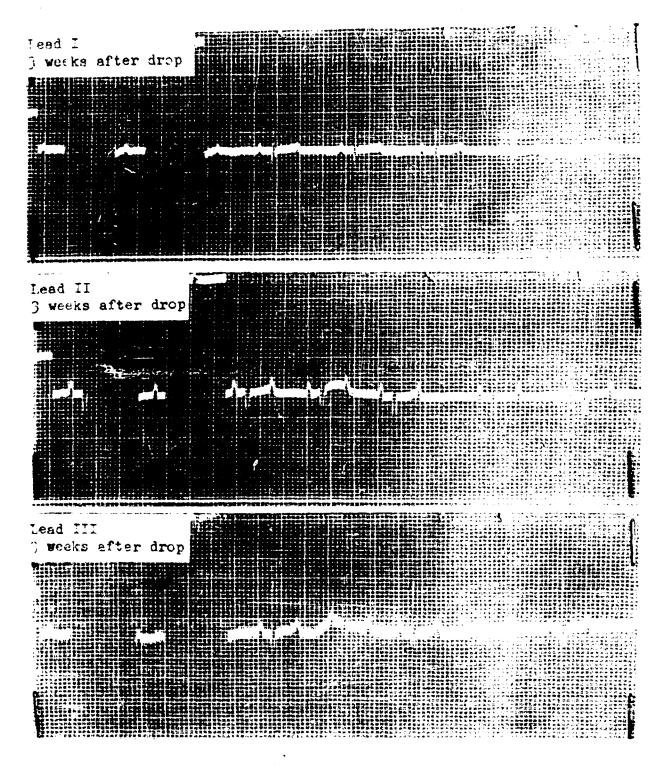


Figure 28. Electrocardiogram of Dog No. 3 three weeks after drop.

EKG Control						Lead III	+ .25 mv	.2 av	1.9 av	o		Dippasic
on None Weight 25 lbs.						Lead II	+ .2 av	.2 mv	1.3 mv	O to slightly depressed	Low takeoff, blends with T wave	
ie Deceleration None	100/min.	Regular	.12 sec.	.03 sec.	.24 sec.	Lead I	+ •1 w	• 3 arv	•5 EV		Slightly elevated (.05)	
Dog Brownie	Heart Rate	Rhythm	P-R Interval	QRS Interval	ORST Interval	1	O.	c c	R	S	ST	

Dog Brownie	Deceieration Approx. 30 g's	ox. 30 g's Weight 25 lbs.	EKG 5 min. after drop
Heart Rate	150/min.		
Rhythm	Regular		
P-R Interval	.ll sec.		
QRS Interval	•04 sec•		
QRST Interval	.20 sec.		
•	Leed 1	Lead II	Lead III
о.	+ .3 tav	+ .25 w	₩ 5° +
0	Ś	o	Inverted
R	1.9 uv	va 6.	1.6 mw (inverted
i,	.2 ev	.15 mv	.3 mv (inverted
ST	Slightly elevated takeoff above control	Slightly elevated takeoff above control	Slightly elevated ta
T.	Diphasic	Diphasic	Diphasic

Dog Brownie Heart Rate	Decele 79/min.	Deceleration	Same	Weight 25 lbs.	lbs.	EXG 55 min. after drop
Irre	Irregular					÷
P-R Interval	.12 sec.					
QRS Interval	.oes to.					
QRST Interval	.28 sec.			<u>-</u> /		
Š	Lead I		T.e	Lead II		Lead III
wa { • • • • • • • • • • • • • • • • • •	•3 av		+	+ .2 mv		.15 wv (inverted)
6	•3 mv		0			.3 mv (inverted)
•	2.2 mv			vm 6.		2.0 mv (inverted)
	.2 uv			.15 av		.l mv (inverted)
ST	ïsoelectric	н С	Elevated take T wave	Elevated takeoff, blends with T wave		Elevated takeoff, blends with T wave
	Dip has ic	н	Diphasic		Ď	Diphasic

A definite sinus arrhythymis is present in all leads. In leads II and III this arrhythemia is present with slight elevation of the ST segment.

Dog Brownie		Deceleration	None	Weight 25 lbs.	EKG 1 hr. 30 min. after drop
Heart Rate	115/min.				
Rhythm	Irregular				
P-R Interval	.10 sec.				
QRS Interval	.04 sec.				
QRST Interval	.20 sec.			ω ^r	•
·	Lead I		Lee	Lead II	Lead III
ρι	÷ •3 av		+	+ .2 mv	+ .15 mv
ð	.25 mv		0		0
R	2.0 mv		`•	·7 mv	лш 6•
8	.2 EV		•	.2 mv	.1 mv
ST	Isoelectric	tric	Slightly above control	ve control	Slightly above control
£1	Diphasic	Ų	Diphasic		Diphasic
					ining the same as the

Sinus arrhythywia still persists with T waves remaining the same as the EKG taken after 55 minutes.

EKG 3 weeks						Lead III	+ .20 uv	.20 mv	1.1 mv	.3 av	Isoelectric
Weight 25 lbs.						I	Ai	A		Į.	tric
						Lead II	+ .25 mv	.20 mv	1.5 000	.35 mv	Isoelectric
Deceleration Same											
Ą	83 /min .	Irregular	.12 sec.	· sas 40.	.25 sec.	Lead I	+ .1 uv	.25 шv	.45 m	.1 mv	Isoelectric
Dog Brownie	Ha.	Rhythm	P-R Interval	ORS Interval	QRST Interval	\$	ė, A	G,	В.	ra :	ST

Sinus arrhythymis still persists.

Upright .25 mv

Upright .3 mv

Upright .15 av

Ę.,

Weight 25 lbs. EKG 11 days	,				
Deceleration Same Wel					
le	79/min.	Irregular	.10 sec.	.05 sec.	.28 sec.
Dog Brownie	Heart Rate	Rhythm	P-R Interval	ans Interval	QRST Interval

Lead TIT	+ O5 my			vm C4.	Slightly elevated above	control Upright .3 mv
Lead II	+ .20 @v	va O4.	1.7 mv	.3 av	Isoelectric (control lead vas	Upright , k mv
Lead I	va + .05 av	20 mv	•• •35 av	·· • • • • • • • • • • • • • • • • • •	· Isoelectric	· Elevated above control
	C ₄	9	æ.	cΩ.	ST	H

Sinus arrhythymia still present with some changes in ST segment and some slight changes in the contour of the T wave.

BIBLIOGRAPHY

I. Nonpenetrating Chest Injury

A. Clinical

- 1. Ahrenberg, H., Traumatic Heart Disease: A Clinical Study of 250 Cases of Nonpenetrating Chest Injuries and their Relation to Cardiac Disability. Annals of Internal Medicine 19:326, 1943.
- 2. Anderson, R. G., Nonpenetration Injuries of the Heart. British Medical Journal 2:307, 1940.
- 3. Barber H., Trauma of the Heart. British Medical Journal 1:433, 1938.
- 4. Barber, H., Electrocardiographic-Changes Due to Trauma. British Medical Journal, 4:83, 1942
- 5. Barber, H., The Effect of Trauma, Direct and Indirect on the Heart. Quarterly Journal of Medicine, 13:137, 1944.
- 6. Barbour, A. B., Injuries in Flying. British Medical Journal, 2:854, 1951.
- 7. Beck, C. S. and Bright, H. F., Changes in the Heart and Pericardium Brought About by Compression of the Legs and Abdomen. Journal of Thoracic Surgery, 2:616, 1933.
- 8. Beck, C. S., Contusions of the Heart. Journal of the American Medical Ass'n., 109: 1935.
- 9. Boas, E. P., Angine Pectoris and Cardiac Infraction from Trauma or Unusual Effort. Journal of American Medical Association 112 1887, 1939.
- 10. Bright, E. F. and Beck, C. S., Nonpenetrating Wounds of the Heart. American Heart Journal, 10:293-321, 1935.
- 11. Foster, R. F., The Relation of Trauma to Heart Disease. Industrial Medicine 8:258, 1938.
- Nonpenetrating Injury to the Chest, Annals of Internal Medicine 27:126 1947.

- 12. Joschim, H., and Mays, A. T., A Case of Cardiac Aneurysm Probably of Traumatic Origin. American Heart Journal, 2: 682. 1926-27.
- 13. Kahn, M. H., and Kahn, B. S., Cardiovascular Lesions Following Injury to the Chest. Annals of Internal Medicine, 2:1013, 1929.
- 14. Kellert, E., Traumatic Rupture of the Heart: A Report of Cases with Uninjured Chest Wall. Journal of Laboratory and Clinical Medicine, 2:726, July 1916-17.
- 15. Kessler, H. H., Accidental Injuries. Philadelphia, Lea and Febiger., 2nd Edition.
- 16. Krumbhaar, E. B., and Crowell, C., Spontaneous Rupture of the Heart. American Journal of Medical Sciences, 170:828, 1925.
- 17. Langendorf, R., and Goldberg, S., The Electrocardiogram in Traumatic Pericarditis. American Heart Journal 24: 412, 1942.
- 18. Leinoff, H. D., Direct Nonrenetrating Injuries of the Heart. Annals of Internal Medicine, 14:653, 1940.
- 19. Leinoff, H. D., Acute Coronary Thrombosis in Industry
 Nonpenetrating Injuries, with Report of Cases. Archives
 of Internal Medicine, 70:33, 1942.
- 20. Masini, V., Alterasioni Miocardiache Nei Traumi Chiusi Del Torace. Rivista Di Medicina Aeronautica, Vol. 11, N., 1, 1948.
- 21. Moritz, A. R., and Atkins, J. P., Cardiac Contusions, An Experimental and Pathologic Study. Archives of Pathology, 24:445, 1938.
- 22. Saphir, Otto, Rupture of the Heart by Indirect Trauma.
 American Journal of Medical Science, 173:353, 1927.
- 23. Schlomka, G., Commotic Cordis und Ihre Folgan, Ergebn.d. inn. Med. u. Kenderh, 97:1, 1934.
- 24. Sigler, L. H., Trauma of the Heart Due to Nonpenetrating Chest Injuries, Journal of American Medical Assn. 119: 855, 1942.

- 25. Sigler, L. H., Traumatic Injury of the Heart. American Journel, 30:459, 1945.
- 26. Smith, L. B. and McKaown, Contusions of the Heart: Report of a Case with Serial Electrocardiogram. American Heart Journal 17:561, 1939.
- 27. Stephens, G. H., Three Thoracic Emergencies. Lancet 2: 1383, December 1922.
- 28. Stromer, A., Traumatic Disease of the Heart. Deutsche Med. Wchnschr, 64:235, 1938.
- 29. Viko, L. E., Communication of 38-year old Man with Symptons of Recent Coronary Occlusion After Being Hit by a Golf Ball Over the Lower Part of the Sternum, 1933.
- 30. Warburg, E., Subacute and Chronic Pericardial and Myocardial Lesions Due to Nonpenetrating Traumatic Injury. Oxford University Press, London, 1938.
- 31. Warburg, E., Myocardial and Pericardial Lesions Due to Nonpenetrating Injury. British Heart Journal, 2:271, 1940.
- 32. Wearn, J. T., In Discussion to Beck, C. S., Contusion of the Heart. Journal of American Medical Association, c.v., 114: 1935.
- 33. Wilson, J.V., The Pathology of Closed Injuries of the Chest. British Medical Journal 1:470, 1943.

B. Experimental

- 34. Kissane, R. W., Fidler, R. A., and Koons, R. A., Electrocardiographic Changes Following External Chest Injury to Dogs. Annals of Internal Medicine, 11:907-935, 1937.
- 35. Kissane, R. W., Fidler, R. S., and Koons, R. A., Traumatic Lesions of the Heart. American Association for the Advancement of Sciences, 13:170-175, 1940.
- 36. Randles, F. S., Gorham, L. W., and Dresbback, M., Changes in the RS-T Component of the Electrocardiogram Produced by Experimental Rupture of the Auricle of Dogs' Heart and by Pericardial Injection. American Reart Journal 9: 333, 1934.

II. Bones

- 37. Calabrisi, P., and Smith, F. C., The Effects of Embalming in the Compressive Strength of a Few Specimens of Compact Human Bone. Naval Medical Research Institute Memorandum Report 51-2 NM OOO 018.07.02 Naval Medical Center, Bethesda, Maryland.
- 38. Carothers, C. O., Smith, F. C., Calibrisi, P., The Elasticity and Strength of Some Long Bones of the Human Body.

 Naval Medical Institute, Project NM OOl 056 13: 1, October 6, 1949.
- 39. Durst, M., Note Sur la Disposition Architecturale due Tissue Spongious des Os: Role de cette disposition dans la production des fractures. Comp Rend et Nemoit de Soc. Ciol, 28:394-387, 1876.
- 40. DeForest, A. V., Ellis, Greer, Brittle Lacquer as an Aid to Stress Analysis. Journal of Aeronautical Sciences 7: 205-208, 1940.
- 41. Evans, F. G., and Lissner, H. R., "Stresscoat" Deformation Studies of the Femur Under Static Vertical Loading. Anat. Rec. 100:159-190, 1948.
- 42. Evans, F. G., Studies of Femoral Deformation. Stanford University Bull, 6:374-381, 1948.
- 43. Evans, F. G., Deformation Studies of the Femur Under Static and Dynamic Loading. Sorbretire de Los Anales del Instituto de Biologia Tomo 20: No. 1y2 Maxico, 1949.
- the Physical Properties of the Human Femur. Journal of Applied Physiology, Vol. 3 No. 9, Merch 1951.
- 45. Evans, F. G., Pedersen, H. E., Lissner, H. R., Role of Tensile Stress in the Mechanism of Femoral Fractures. Journal of Bone and Surgery, Vol. 33-A, No. 2, 485-501, April 1951.
- 46. Evans, G., and Lebow, Milton, Strength of Human Compact Bone as Revealed by Engineering Techniques. American Journal of Surgery, Vol. 83: No. 3 326-331, 1952.
- 47. Evans, G., and Lessner, H. R., Deformation Studies of the Adult Human Pelvis Under Dynamic Loading. Dept. of Engineering Mechanics, Wayne University, 1953. (Meeting of American Association of Anatomists).

- 48. Fasola, Alfred, Anatomical and Physiological Effects of Rapid Deceleration. Master's Thesis Ohio State University, 1950.
- 49. Fick, R., Handbuch der Anatomie und der Gelenke Dritter Teil Spesielle Gelenl-und Muskelmechnik, Gustav Fishher, Jena.
- 50. Gurdjien, E. S., and Lissner, H. R., Deformation of the Skull in Head Injury, A Study with the "Stresscoat" Technique.

 Surgery, Gynecology, and Obstetrics, 81:679-687, 1945.
- 51. Gurdjian, E. S., and Lissner, H. R., Deformation of the Skull in Head Injury Studied by the "Stresscoat" Technique, Quantative Determinations. Surgery, Gynecology, and Obstetrics, 83: 219-233, 1946.
- 52. Gurdjian, E. S., Lissner, H. R., and Webster, J. E., The Mechanism of Production of Linear Skull Fracture. Further Studies on Deformation of the Skull by the "Stresscoat" Technique. Surgery, Gynecology and Obstetrics 85:195-210, 1947.
- 53. Gurdjian, E. S., and Lissner, H. R., Deformation of the Skull in Head Injury as Studied by "Stresscoat" Technique. American Journal of Surgery 73:269, 1947.
- 54. Evans, F. G., Lissner, H. R., and Pedersen, H. E., Deformation Studies of the Femur under Dynamic Vertical Loading.

 Anat. Rec. 101:225-241, 1948.
- 55. Gurdjian, E. S., and Lissner, H. R., Anatomy and Some Principles of Mechanics. (College of Engineering) Wayne University, Anat. Rec. Volume No. 3, March 1949.
- 56. Gurdjian, E. S., Webster, J. E., and Lissner, H. R., Studies with Particular Reference to Engineering Factors. American Journal of Surgery 78:736-742, 1949.
- 57. Gurdjian, E. S., Webster, J. E., and Lissner, H. R., Mechanism of Skull Fracture. Journal of Neurosurgery, 7: 106-114, March, 1950. Radiology 54:313, March, 1950.
- 58. Jones, E. S., Joint Lubrication. Lancet 1:1043-1044, 1936.
- 59. Koch, J. C., Laws of Bone Architecture. American Journal of Anatomy 21:177-298, 1917.
- Maj, C., and Toaiari, E., Caservazoni sperimentali sul Meccaismodi Resistenza del Tessuto Osseo Lamellare Compatto alle azioni Meccanchi. Chir.d. org di Movimento 22:541, 1937.

- 61. Maj, G., Monit 2011 Ital 49:139, 1938.
- 62. Maj, G., Resistenza Mecchanica del Tessuto Osseo e Diversi Livelli di Uno Stesso Osso. Boll. Soc. Ital, Sper. 13: 413-415, 1938.
- 63. Maj, G., Variasioni Individuali e Topographicle dell Resistenza Mecchanica del Tessuto Osseo Umano. Boll. Soc., Ital, Ciol. Sper 15:1151, 1940.
- 64. Maj, G., Studio sulle Varioasioni Individuali e Topographiche delle Resistenza Mecchanica del Tessuto Osse Diafeiario Umano in Diversi eta. Arch. Ital, Annat e Embriol 47:612, 1942.
- 65. Marique, P., Etudes sur le Femur. Bruxcelles, Belgium: Librairel des Sai.
- 66. Messerer, O., Eber Elasticitat und Festigkeit der Menschl Chen Knochen. Stuttgart, Germany Cotta 1880.
- 67. Milch, H., Photoelastic Studies of Bone Form. Journal of Bone and Joint Surgery, (N.S.) 22:621-626, 1940.
- 68. National Bureau of Standards; Compressive Tests of Tubular Specimens of Human Bone Report No. 2, U.S. Dept. of Commerce, Naval Medical Institute, Bethesda, Maryland, August 13, 1948.
- 69. National Bureau of Standards; The Mechanical Properties of Bones; U.S. Dept. of Commerce, Progress Report No. 1, NBS, Lab. 64219, Washington, D.C., January 31, 1948.
- 70. National Bureau of Standards; Tensile Tests of Specimens
 Removed from Human Femurs. Naval Medical Research Institute,
 Bethesda, Maryland, January 27, 1949.
- 71. Pedersen, H. E., Evans, F. G., Lissner, H. R., Deformation Studies of the Ferur under Various Loadings and Orientations. Anat. Rec. 103:
- 72. Rauber, A. A., Elasticitat und Festigkeit der Knochen. Leipzig, Englemenn, 1876.
- 73. Spears, G. M., Owen, J. T., The Etiology of Trochanteric Fractures of the Femur. Journal of Bone and Joint Surgery, 31-A: 548-552, July, 1949.
- 74. Toaiari, E., Resistenza Meccanica e Elasticita del Tessuto Osseo Studiata in Rapporto all Minuta Strutura. Monit. Zool. Ital 47:148-154, 1938.

- 75. Tomiari, E., Monit, Zool. Ital, 48:148, 1938.
- 76. Walmsley, T., The Vertical of the Femur and their Relations.
 A Contribution to the Study of Erect Posture. Journal of
 Anatomy 67:284-300, 1932.
- 76a. Wolf, Julius, Das Gesetz der Transformation der Knochen. Onarto, Berlin 1892.

III. General Aspects of Daceleration

- A. Exposure to long "g" deceleration
 - 77. Beckman, E. L., Protection Afforded Cerebrospinal System by Cerebrospinal Fluid Under Stress of Negative G.
 Journal of Aviation Medicine, 20:430, December, 1949.
 - 78. Christy, R. L., New Human Centrifuge. Journal of Aviation Medicine, 20:454-458, December, 1949.
 - 79. Denzin, E. C., Decelerator for Human Experimentation.
 A. F. Tech. Report, U.S. Air Force (5973) pp 1-70,
 February, 1950.
 - 80. Gamble, J. L., Jr., and Shaw, R. S., Pathology in Dogs Exposed to Negative Acceleration. Air Materiel Command Memorandum Report No. TSEAA-095-74B, August, 1947.
 - 81. Gamble, J. L., Jr., and Shaw, R. S., Gauer, C. H., and Henry, J. F., On Cerebral Dysfunction During Negative Acceleration. Journal of Applied Physiology, 2:133, 1949.
 - 82. Henry, J. P., Gamble, J. L., Shaw, R. S., Gauer, O. H., Martin, E. E., Maher, P., Jr. and Simmons, D. G., Studies of the Physiology of Negative Acceleration. USAF Technical Report No. 5953, October 1950.
 - 83. Lombard, C. F., Beckman, E. L., Rushmer, R. F., Drury, D. R., Goodman, H., and Edmonson, H., Effects of Radial Acceleration on Large Experimental Animals (goats).

 I-Pathology. Office of Naval Research Contract No. 60 re 77, Project NR 161014, November, 1948.
 - 84. Lambert, C. H., Comparison of the Protective Value of Anti-Blackout Suit on Subjects in Airplane and Mayo Centrifuge. Journal of Aviation Medicine, 21:28-37, February, 1950.

- 85. Heary, J. P., Use of the Anti-G Suit to Aid in the Relief of Fighter Pilot Discomfort. Memorandum Report, TSEAA 680-2a, Wright-Patterson Air Force Rase, September 22, 1947.
- 86. Henry, J. P., Gambel, J. L. Jr., Shaw, R. S. and Gauer, O. H., Venous Pressure in the Head Under Negative Acceleration. Memorandum Report, MCREXD-695-74-A, Wright-Patterson Air Force Base, Dayton, Ohio, June 16, 1948.
- 87. Henry, J. P. and Shaw, R. S., The Significance of the Volume of Blood Contained in the Legs in Negative Acceleration. Memorandum Report, MCREXD-695-74-J, Wright-Fatterson Air Force Base, Dayton, Ohio, May 11, 1948.
- 88. Martin, E. E., Service Test Report on USAF Type G-4A
 Pilots Pneumatic Suit, Anti-G, Memorandum Report,
 MCPEXD-589-2E, Wright-Patterson Air Force Base, Dayton,
 Ohio, May 20, 1949.
- 89. Mertin, E. E. and Whitney, R. W., A Description of the U.S. Air Force Type M-7 No-Leak Negative 'g' Valve. WADC Technical Note No. 52-11.
- 90. Rushmer, R. F., Beckman, E. L., and Lee, D., Frotection of the Cerebral Circulation by the Cerebrospinal Fluid Under the Influence of Negative 'g'.

 American Journal of Physiology, 151: 355, 1948.
- 91. Ryan, E. A., Kerr, W. K., and Franks, W. R., Some Physiological Findings on Normal Man Submitted to Negative Acceleration. Report RCAF, Institute of Aviation Medicine, Toronto, Canada, 1948.
- 92. Shaw, R. S., and Henry, J. P., The Pressurized Helmet as a Negative 'g' Protective Device. Air Materiel Command Memorandum Report TSEAA-660-100, App. May 4, 1946.
- 93. Sieker, H. O., Capt., Devices for Protection Against
 Negative Acceleration. WADC Technical Report 52-67,
 Part I, Wright-Patterson Air Force Base, Dayton, Ohio.
- 94. Simons, D. G. Henry, J. P., Electronencephalographic Changes Occurring During Negative Acceleration (Headward Centrifugal Force), AF Technical Report No. 5966, Wright-Patterson Air Force Base, Dayton, Ohio, May, 1950.

95. Wood, J., Cain, C. C., and Makoney, D. I., Physiological Evaluation of the Partial Pressure Suit.

Air Materiel Command Report MCREXD-696-104P (unpublished).

B. Exposure to Impact 'g'

- 96. All American Aviation, Research Progress of Unolyn (Impact Absorption Material to May 15, 1946).
 Wilmington, Delaware, May 24, 1946.
- 97. Reference deleted.
- 98. Penrod, K. E., Comparison of Opening Shock Forces and Descent Times of 24' and 28' Nylon Parachute at Various Altitudes. May 22, 1945. Memorandum Report, TSEAL3-696-666, Wright Field, Dayton, Ohio.
- 99. Maison, G. L., Descent Times of 200 lb. Dummies with 24' and 28' Nylon and 28' Silk Parachutes Opened at Various Altitudes. May 30, 1945. Memorandum Report TSEAL3-696-66H. Wright Field, Dayton, Ohio
- of Ripstop Nylon and Standard Nylon Parachutes,
 Memorandum Report No. TSEAL3-696-661, 20 July 1945,
 Wright Field, Dayton, Ohio
- 101. Report No. 1, SAM, Army Air Force, Internal Injuries
 Produced by Abrupt Deceleration of Experimental
 Animals. Project 401 January 15, 1940.
- 102. Army Air Force, Effects of Abrupt Deceleration on the Electrocardiogram (Lead II) in the Cat in the Supine Position. Report No. 1 SAM, Project No. 459 January 21, 1946.
- 103. Aero Medical Research News Letter, Investigation of Human Tolerance to Parachute Opening. Aeronautical Medical Equipment Laboratory, BU Med Project NM 001 062.03, Progress Report, March, 1951.
- 104. Bierman, H. R., and Larsen, V. R., Reactions of Humans to Impact Forces Revealed by High Speed Motion Ficture Technique. Journal of Aviation Medicine, 17: 407, 1946.

- 105. Cornell Aeronautical Laboratory, Inc., Kinematic Behavior of the Human During Crash Deceleration (Thin Men Project). Internal Research Project, Report No. OM-596-J-1.
- 106. DeHaven, H., Crash Injury Research, Crash Deceleration, Crash Energy, and Their Relationship to Crash Injury. Technical Report AFTR-6242, 1950.
- 107. Hallenback, G. A., The Magnitude and Duration of Parachute Opening Shocks at Various Altitudes and and Air Speeds. Memorandum Report ENG-49-696-66, Wright Field, Dayton, Ohic, 8 July 1944.
- 108. Joffee, M., Anatomical and Physiological Effects of Abrupt Deceleration. Ph.D. Dissertation, Obio State University, 1949.
- 109. Leavell, B. S., Acute Heart Failure Following Blast Injury. War Medicine 7: 162, 1945.
- 110. Shaw, R. S., Human Tolerance to Negative G Short Duration. AMC Memorandum Report, TSEAA-695-74, Wright Field, Dayton, Ohio, May 19, 1947.
- 111. Gamble, L. L. Jr., Animal Studies in Impact Negative Acceleration. AMC Memorandum Report, MCREXD-595-74-G Wright-Patterson Air Force Base, Dayton, Ohio. March 10, 1948.

ومدور والمرامية ممك مطاملته وقيم معلمات عاقما والقلاف سيلمه والأقامة وميدو هم ووسائطة المسامة المعارمة

- 112. Ruff, S., Concerning Human Tolerance of Acceleration as it Applies to Certain Jerking Types of Acceleration Which Occur in Flying. (Presented at the German Academy of Aviation Development on October 31, 1941. Translated in Crash Injury Research Report on September 27, 1947) 1941.
- 113. Ruff, S., Brief Acceleration; Less Than One Second. German Aviation Medicine, World War II, Vol. 1, Chapter VI-V, p. 584-586.
- 114. Shaw, R. S., Acceleration Time Diagrams for Catapult Ejection Seats. AMC Memorandum Report No. TSEAA-695-66D, Wright Field, Dayton, Ohio, February, 1947.
- 115. Shaw, R. S., Test Firing of T-D Catapult for Downward Ejection. AMC Memorandum Report No. MCREXD-695-74- I, Wright Field, Dayton, Ohio, May, 1948.

- 116. Stapp, J. P., Human Exposure to Linear Deceleration:
 Preliminary Survey of Aft-Facing Seated Position,
 A.F. Technical Report No. 5915, Part I, U.S. Air
 Force, June, 1949.
- 117: Webster, A. P., National Advisory Committee for Aeronautics, Free Falls and Parachute Descent in Standard Atmosphere. Bureau of Medical and Surgery, Naval Department.

C. Miscellaneous.

- 118. Armstrong, H., and Heim, J. W., Effect of Acceleration on the Living Organism. USAAF Technical Report No. 4362, December, 1937.
- 119. Aviation Week, Crash Safety Can Be Engineered. McGraw-Hill Publishing Co., New York 18, N.Y., March 13, 1950.
- 120. Bierman, H. R., Wilder, R., Jr., Hellems, H., The Principles of Protection of the Human Body as Applied in a Restraining Harness for Aircraft Pilots. Naval Medical Research Institute, Bethesda, Maryland.
- 121. Crash Injury Research, Handbook for Aircraft Accident Investigators. Cornell University Medical College.
- 122. Crash Injury Research, Dept. of Commerce, Civil Aeronautics Authority, 612.1 (03.38) Washington, D.C., July 1, 1948.
- 123. Crash Injury Research, Hazard of Seat Back in Aircraft Accidents. National Research Council, October 18, 1946.
- 124. Crash Injury Research, Informative Accident Release, January 1, 1947.
- 125. Crash Injury Research, The Rare Occurrence of Internal Abdominal Injury from Safety Belts or Other Causes in Serious Aircraft Accidents. October, 1947.
- 126. Crash Injury Research Release, The Function of Safety Belts in Crash Protection. February 20, 1948.
- 127. Crash Injury Research, Informative Accident No. 7, National Research Council, May 7, 1948.

- 128. Crash Injury Research, Informative Accident No. 8, Cornell Committee for Air Safety Research, Cornell Medical College, September 15, 1949.
- 129. Crash Injury Research, Human Tolerance to Deceleration. Committee for Air Safety Research, Cornell Medical College, January 31, 1950.
- 130. Crash Injury Research, Informative Accident No. 9,
 National Research Council, Cornell Medical College,
 May 18, 1950.
- 131. Crash Injury Research, Informative Accident Release
 No. 11, National Research Council, Cornell Medical
 College, April 25, 1951.
- 132. Crash Injury Research, Informative Release No. 13, Cornell Medical College, August 30, 1951.
- 133. Crash Injury Research, The Site, Frequency and Dangerousness of Injury Sustained by 500 Survivors of Light-Plane Accidents. Dept. of Public Realth and Preventive Medicine, Cornell University Medical College, July, 1952.
- 134. Crash Injury Research, Crash Injury Study of the Northeast Airlines Conveir 240 Accidents at Laguardia Airport on January 14. Informative Accident Release No. 14, August, 1952.
- 135. Crash Injury Research, Shoulder Harness, Its Use and Effectiveness, Dept. of Fublic Realth and Preventive Medicine, Cornell University Medical College, November 1, 1952.
- 136. DeHaven, H., Mechanical Analysis of Survivable Accidents in Falls from Heights of 50 to 150 Feet. War Medicine Vol. 2, p 586, 1942.
- 137. DeHaven, H., Injuries in Thirty Light-Aircraft Accidents. Report No. 230, Committee on Aviation Medicine, National Research Council, November 17, 1943.
- 136. DeHaven, H., Causes of Injury in Light-Plane Accidents. Aero Digest, Vol. 44, 51, 1944.
- 139. DeHaven, H., Crash Research from the Point of View of Cabin Design. Aeronautical Engineering Review, Vol. 4 No. 6, June 1946.

- 140. DeHaven, H., Crash Injury Research, Semi-Annual Progress Report, Cornell Medical College, January 31, 1952.
- 141. DeHaven, H., Crash Injury Research, Current Safety
 Considerations in the Design of Passengers Seats
 for Transport Air-craft. Cornell University,
 Medical College, June, 1952.
- 142. DuBois, E., Limits of Factors of Safety in the Human Body. Mechanical Engineering, July, 1946.
- 143. German Aviation Research Report No. 1549 P395 T 7; Seat Installation with Steering for High Flying Acceleration - written by German Research Institute for Aviation, E. Berlin.
- 144. Hertzberg, H. T. E., and Colgan, J. W., A Prone Posttion Bed for Pilots. Air Materiel Command, Memorandum Report MCREXD-695-71D June, 1948.
- 145. Limits and Special Problems in the Use of Seat Catapult. approximately 1946 (author unknown)
- 146. Martin, E. E., Evaluation of the Anti-G-Suit, Report
 No. 8. Memorandum Report MCREXD-689-2C, WrightPatterson Air Force Base, Dayton, Ohio, July 24, 1948.
- 147. National Research Council, Committee on Aviation Medicine, The Relationship of Injuries in Survivable Aircraft Accidents. Report No. 440, July 9, 1945.
- 148. Research on Crash Injury. Journal of American Medical Association, June 8, 1946.
- 149. Schneider, J., Protective Measures for Prevention of Injuries; Especially Spinal Fractures in Aircraft on Skids. German Aviation Medicine, World War II, Vol. 1, p 612.
- 150. Stapp, J. P., Human Exposures to Linear Deceleration.
 Part 2. The Forward-Facing Position and the Development of a Crash Harness. Air Force Technical Report
 5915, Wright-Patterson Air Force Base, Ohio, December
 1951.
- 153. Teare, D., Analysis of the Viking Aircraft Accident. British Medical Journal 2: 1337, 1951.

Measurements of the Loads Required to Break Commercial Aviation Safety Belts as an Indication of the Ability of the Human Body to Withstand High Impact Forces.

Report No. 12, Project NM 001 006 X-630 - Naval Medical Research Institute, National Medical Center, Bethesda, Maryland.